

DOES NOT CIRCULATE

PUBLISHED WEEKLY



PRICE TWO SHILLINGS
AND SIXPENCE OF MICHIGAN

OCT 20 1954

MEDICAL
LIBRARY

THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—41ST YEAR

SYDNEY, SATURDAY, SEPTEMBER 11, 1954

No. 11



for both Children and Adults

DOSE

ADULTS: One tablespoonful
three times a day.

CHILDREN: Half to one
teaspoonful three times a
day

Vimalt provides in a
pleasant orange flavour malt base
Vitamins A, B₁, and D, and Iron.

EACH OUNCE CONTAINS

Vitamin A	- - -	5,000 I.U.
Vitamin B ₁	- - -	200 I.U.
Vitamin D	- - -	500 I.U.



F. H. FAULDING & CO. LTD.

MANUFACTURING CHEMISTS — AUSTRALIA

F. H. F. 3

ACTH-DEPOT

SCHERING A.G. BERLIN

The
adrenocorticotrophic
hormone preparation
of high purity

Prolonged action of at least 24 hours

Multidose vials of 5 cc. containing 100 I.U. and 200 I.U. each

SCHERING A.G. BERLIN

Distributors:

SCHERING PTY. LTD., 52 CARRINGTON ST., SYDNEY

THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—41ST YEAR

SYDNEY, SATURDAY, SEPTEMBER 11, 1954

No. 11

Table of Contents.

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES—	Page.	ON THE PERIPHERY—	Page.
Poliomyelitis: The Experimental Approach to its Prevention, by N. F. Stanley ..	417	An Account of State of Health and Problems of Medical Interest in Gollala, Papua ..	444
An Approach to Neuroses in General Practice, by M. G. Jansen ..	422	BRITISH MEDICAL ASSOCIATION NEWS—	
An Electrolyte Conversion Table, by J. A. C. Dique ..	428	Scientific ..	447
Post-Gastrectomy Dietary Management, by Patricia J. Crowe and Stanley Goulston ..	430	OUT OF THE PAST ..	452
The Pattern of Congenital Heart Disease in Infancy and Childhood, by Douglas Stuckey ..	433	SPECIAL CORRESPONDENCE—	
REVIEWS—		Paris Letter ..	452
Diseases of the Knee ..	434	CORRESPONDENCE—	
Anatomy for Surgeons. Volume I: The Head and Neck ..	435	Medical Aspects of Tattooing ..	454
Diseases of the Liver, Gallbladder and Bile Ducts ..	435	The Enigma of the Mona Lisa Smile ..	454
Metabolic and Toxic Diseases of the Nervous System ..	435	Concerning Proctology ..	454
Annals of Medical Detection ..	436	NAVAL, MILITARY AND AIR FORCE—	
The Year Book of the Eye, Ear, Nose and Throat ..	436	Appointments ..	455
The Year Book of Orthopedics and Traumatic Surgery ..	436	DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA ..	455
The Bible and Modern Medicine ..	436	AUSTRALIAN MEDICAL BOARD PROCEEDINGS—	
BOOKS RECEIVED ..	436	Queensland ..	456
LEADING ARTICLES—		Tasmania ..	456
Trends in Nursing ..	437	NOTICE—	
CURRENT COMMENT—		Australian Association of Clinical Pathologists ..	456
Adrenalectomy in Relation to Adrenal Tumours ..	438	MEDICAL APPOINTMENTS ..	456
Abraham Lincoln: Medical Aspects of His Assassination ..	439	DEATHS ..	456
Hypertensive Encephalopathy and its Relation to the Malignant Phase of Hypertension ..	440	DIARY FOR THE MONTH ..	456
Men, Women and Types of Motor-Car Accidents ..	441	MEDICAL APPOINTMENTS: IMPORTANT NOTICE ..	456
ABSTRACTS FROM MEDICAL LITERATURE—		EDITORIAL NOTICES ..	456
Dermatology ..	442		
Urology ..	443		

POLIOMYELITIS: THE EXPERIMENTAL APPROACH TO ITS PREVENTION.¹

By N. F. STANLEY,

Acting Director, Institute of Epidemiology and Preventive Medicine, The Prince Henry Hospital, Sydney.

THE most hopeful approach for the immunization of humans against poliomyelitis surely lies in the production of a living, attenuated virus vaccine, comprising the three antigenic types of poliomyelitis and administered orally. This view is definitely favoured by Cox (1953), and probably by Burnet (1951), Theller (1952) and Sabin (1953). The factors which lead me to make the foregoing statement provide the material for this paper.

In studying the problems associated with the prevention of poliomyelitis epidemics, both epidemiology and pathogenesis should be considered. The epidemiological picture is by no means static, and this changing panorama was recently summarized by John Paul at the Second International Poliomyelitis Congress and quoted by Horstmann (1953) as follows:

¹Read at a meeting of the Section of Pathology and Bacteriology of the New South Wales Branch of the British Medical Association on May 11, 1954.

A disease originally regarded as being limited to infants is now no longer confined to infancy. A disease originally considered as mildly contagious is now regarded as very contagious—almost as much so as measles. A disease in which the clinical picture was originally thought to be limited to acute paralysis now is regarded as a disease in which only 1 in 100 or more of those infected become paralysed—and if the Lansing strain is involved, perhaps 1 in 1000. From an endemic disease it has tended to become epidemic and is now a common and periodic scourge.

Very few epidemiological problems have been solved. Most experimental work supports the observation that virus is spread by close human contact, but does not explain the seasonal incidence. The large number of cases occurring in the summer and early autumn is probably associated with a greatly increased dissemination of the virus in the community; but the possibility also exists that there is a decrease of resistance on the part of the host. This latter suggestion receives some support from quantitative studies on the antibody content of pooled human serum in the Sydney metropolitan area (Stanley, unpublished observations). Furthermore, we have no satisfactory explanation for the varied clinical manifestations of poliomyelitis infection, ranging from inapparent infection and non-paralytic infection, through paralysis to death. Then there is the ratio of inapparent infections to clinical cases—such a ratio apparently showing considerable variation

with the type of virus involved, the location of the epidemic and the state and age of the host. Burnet (1945) and Sabin (1951) hold that the inapparent infection rate is greater in infants than in older children—which means (as Horstmann suggests) that older children are more susceptible to paralysis than younger ones.

One of the major changes that has occurred since 1870, when poliomyelitis was first recognized in epidemic form, is the steady rise in age incidence, which is particularly pronounced in Australia, the Scandinavian countries and the United States of America. In these countries it is no longer an infantile disease. An interesting feature about this increase is that it has not occurred everywhere. Poliomyelitis is still "infantile paralysis" in Cuba, Panama and Israel (where recent epidemics have occurred), as well as in Egypt, where no epidemics occur in the native population (Horstmann, 1953). We cannot explain this phenomenon; but it has been suggested on several occasions that the change is related to the introduction of modern hygiene and high standards of living, which have resulted in the protection of children from exposure to virus at an early age with the subsequent building up of an ever-increasing number of susceptibles.

Both Hammon and his associates (Hammon *et alii*, 1950) and Melnick and Ledinko (1951) have produced data which suggest that different patterns of susceptibility may occur even in one city. They did this by comparing the results of antibody estimations (Lansing type) from "upper and lower socio-economic groups". These observations lend support to the hypothesis that populations living under primitive sanitary conditions become exposed to infection at an early age and the disease remains endemic. Horstmann (1953) quotes the now classical examples of Miami and Cairo. Both are in the same latitude, but in Cairo antibodies appear very early together with the clinical disease, whereas in Miami antibodies appear much more slowly and clinical poliomyelitis develops at a later age.

One of the main topics for disagreement as far as pathogenesis is concerned is whether the virus is a strictly neurotropic agent, or whether there is a primary non-neural development and possibly blood-borne infection. At the present time no conclusive data are available. We know that the virus grows well in non-nervous tissue, and may be easily recovered from the gastro-intestinal tract of man, from raw sewage and occasionally also from the blood. The bulk of evidence suggests to me that it is primarily a gastro-intestinal parasite.

It is certain that much of this confusion about poliomyelitis has arisen because competent laboratory tests were not devised for the rapid and accurate detection of virus or antibody to it. Our resulting ignorance has, until recently, almost completely invalidated the studies on immunization that were carried out between 1920 and 1950. It is useless to produce a vaccine if satisfactory methods for testing it are not available. The methods have only just been devised and are being improved. It is obvious that progress in the prevention of poliomyelitis is going along hand in hand with technical advance and is dependent on it. There appear to be no new principles involved, just the simple application of a few technical tricks in studying the pathogenesis of the disease under varying conditions in different hosts.

The whole problem of preventing epidemics of poliomyelitis must surely be directed (at this stage) toward maintaining a certain level of antibody in a certain percentage of the population. While discussing the means at our disposal to bring about this state of affairs, I hope also to illustrate the important part technique has played.

There are two methods of immunization—passive and active.

Passive Immunization.

Passive immunization has been tried on humans in the United States of America on a large scale and at considerable expense. The substance used was the γ globulin fraction of pooled adult serum, and it was used largely at

the instigation and under the direction of Dr. W. McD. Hammon. A large number of scientists, including Dr. Hammon, are now in agreement that γ globulin therapy is of little practical value in preventing poliomyelitis epidemics. It has some limited use as a stop-gap until a suitable vaccine becomes available and will possibly be of value in temporarily protecting household contacts. However, the γ globulin experiments did reveal at least one thing of value, and that was the low level of serum antibody required to confer immunity on a human being (Hammon, 1953). Until the first γ globulin experiments in Utah in 1951, there was no animal experimental work on record which suggested that protection was forthcoming by anything other than very high levels of serum antibody. These tests showed that temporary protection was afforded by administering a limited dose of γ globulin, and the amount of circulating antibody would probably not even be detected by the current methods of testing. Again we are technically at fault in not having sensitive enough tests for antibody. The tests also showed that artificial laboratory experimental procedures in animals by no means duplicated the state of affairs existing by the natural exposure of the natural human host (Hammon, 1953).

According to Cox (1953):

If an economically practical method for determining susceptibility to poliomyelitis were available then the use of immune serum-globulin might be justifiable; but as we lack the necessary diagnostic tests, a public-health programme which utilizes this method of prophylaxis must recognize that the limited supply of gamma globulin may be needlessly dissipated in children who are already immune to poliomyelitis.

Active Immunization.

Without doubt, then, the obvious approach to the problem of the prevention of poliomyelitis is to devote considerable time and energy to the development of a vaccine—one that will, if possible, give a long-lasting immunity. There are two possibilities—a killed vaccine or a living attenuated virus vaccine. I propose now to point out some of the advantages and disadvantages of each type.

Killed Virus Vaccines.

First let us consider the killed poliomyelitis virus vaccines. These are many and varied, but none have yet proved to be successful. These have been treated by various physical agents, such as the following: (i) drying (Levaditi and Landsteiner, 1910; Pollard, 1951); (ii) heat (Abramson and Gerber, 1918; Shaughnessy *et alii*, 1930; Melnick, 1951); (iii) ultrasonic treatment (Kasahara, 1939); (iv) ultraviolet irradiation (Milzer *et alii*, 1945; Morgan *et alii*, 1947; Dick *et alii*, 1951; Milzer *et alii*, 1954); (v) electron bombardment (Dick *et alii*, 1951). However, none have proved to be superior in monkeys to the preparations treated with chemicals.

Some of the chemicals used have been the following: (i) phenol (Aycock and Kagan, 1927; Cowie, 1935; Brodie, 1938); (ii) aluminium hydroxide (Rhoads, 1930, 1931; Sabin, 1931); (iii) ricinoleate (Kolmer, 1938); (iv) formalin (Brodie, 1935).

Kolmer's ricinoleate-treated vaccine was given subcutaneously to 10,752 persons in 1935, and nine cases of paralytic poliomyelitis developed in subjects who had received it. At the same time Brodie's formalin-treated vaccine was tried in humans. Although this was not followed by cases of paralysis, an insufficient number of persons was inoculated to determine its efficacy. Trask and Paul (1936) showed that formalin (0.1%) did not destroy the virus after twenty-four hours at 4° C., but Brodie's vaccine was allowed to react with formalin (0.1%) for only eight to twelve hours. Some doubt, therefore, existed as to its avirulent nature. The ricinoleate preparation was shown to contain live virus. The use of both vaccines was therefore stopped. Both preparations contained mixed antigenic types, and at the time very little was known about the three types as we know them today. The results are inconclusive.

Morgan (1948) showed that repeated inoculation of monkeys with a formalinized vaccine produced demonstrable immunity. Four years later, Howe (1952) reported the results of treating chimpanzees and humans with a formalinized preparation of spinal cords of infected monkeys. Cox (1953) stated that such vaccines might be immediately dismissed from practical consideration, because (i) poliomyelitis virus could not be concentrated and readily freed of the nervous tissues associated with allergic encephalitis, and (ii) it was doubtful whether there were sufficient monkeys available to produce such a vaccine commercially. These difficulties may shortly be overcome. For example, the contributions of Enders and his associates (Enders *et alii*, 1949; Weller *et alii*, 1949; Robbins *et alii*, 1951) in showing that poliomyelitis viruses can be cultivated in tissues of non-neural origin are of outstanding importance in this respect and remove the risk of allergic encephalitis. Tissue culture is also of undoubted importance as a method of detection and assay, as well as for the preparation of diagnostic antigens and vaccines.

Finally, Salk (1953) has reported preliminary experiments with formalin-treated preparations derived from tissue cultures. In order to detect antibodies against all three types, it was found necessary to use an adjuvant (water in mineral oil emulsion). Antibody titres were reported on only a short time after vaccination, and most of the people were found to have antibodies prior to testing. (In fact, all had antibodies against type I before the vaccine was administered.) Cox (1953) is critical of these publications and suggests the possibility that malignant tumours might occur in susceptible persons as a result of mineral oil given subcutaneously. Cox also indicates that it may be necessary to revaccinate the subject every two or three years in order to maintain antibody to the level required for protection. Some of the disadvantages of the killed virus vaccine, therefore, are as follows: (i) irritation to the tissues by the chemical constituents following subcutaneous inoculation; (ii) allergic encephalitis (depending on the source of the material); (iii) dangers resulting from use of adjuvants; (iv) repeated vaccination of children every few years—resulting in sensitization of the subject to some foreign product in the vaccine; (v) the preparation of large quantities of purified vaccine. Cox holds that it will take much longer to evaluate the safety of a mineral-oil-adjuvant type vaccine than that of a living, attenuated virus vaccine.

Living Virus Vaccines.

Without any doubt, the most successful virus vaccines have so far been living ones. The successful living vaccines in humans are those of smallpox, yellow fever and rabies. Of considerable interest for a comparison with poliomyelitis is the yellow fever vaccine. Two types are being used: (i) the chick-embryo-propagated 17D vaccine of Theiler (1951, 1952), and (ii) the French neurotropic vaccine.

These have their origin in the twenty-year-old observation of Theiler (1930) that the yellow fever virus could infect white mice by the intracerebral route. This process resulted in some loss of virulence for monkeys. In the French territories of Africa, the mouse-adapted strain is extensively used by scarification of skin with a dried infected mouse-brain preparation (Peltier *et alii*, 1940). Theiler's chick-embryo propagated vaccine is injected subcutaneously, and consists of reconstituted dried infected chick-embryos. (This presumably imitates the mosquito-bite—the natural route of infection for man.) The vaccine induces immunity by producing an infection, as with all living virus vaccines. Millions of humans have been vaccinated with the 17D vaccine, and since it has been introduced there have been no accidental laboratory infections, which were once so common.

On the veterinary side, success has attended the determined and persistent attempts of Cox and his associates with (i) Newcastle disease virus (Markham *et alii*, 1951), (ii) rabies (Koprowski and Cox, 1948)—this was chick-embryo propagated, (iii) canine distemper (Cabasso and

Cox, 1949)—also chick-embryo propagated, and (iv) hog cholera, rabbit adapted (Koprowski *et alii*, 1952). There has never been an indication that any of these attenuated living virus vaccines revert to their original virulence or transmissibility (Cox, 1953). Other successful live vaccines are those of rinderpest, South African horse sickness, blue tongue of sheep, fowl pox and infectious laryngotracheitis of chickens.

The use of infected mammalian tissues as a source of live virus for immunizing is subject, in large-scale work, to the risk of contamination with latent viruses such as that of lymphocytic choriomeningitis, encephalomyocarditis virus, Sabin's B virus, *et cetera*. This, and other factors, led Cox to believe that the developing chick embryo, which is considered to be free from latent virus, could be of very great value in propagating poliomyelitis viruses for the production of a live vaccine. The developing chick embryo has been used successfully for the large-scale production of living virus vaccines for yellow fever, smallpox, rabies, blue tongue of sheep, rinderpest, Rift Valley fever, Newcastle disease virus, canine distemper and fowl pox (Cox, 1953).

I wish to quote Herald Cox in detail from a recent paper, in which he states that Newcastle disease vaccine has served as a very useful model for much of his colleagues' investigations on a live poliomyelitis vaccine. It was shown:

... that by using the proper quantitative conditions a single drop of the relatively avirulent Blacksburg strain of Newcastle disease virus will readily immunize either fully susceptible, or passively immune, one-day-old baby chicks, provided the portals of entry of natural virus infection, namely the intranasal or conjunctival routes, are used. Such vaccinated birds show no illness following vaccination and often fail to develop any appreciable antihæmagglutinins, particularly if the virus menstium possesses no unusual surface activity; yet they will readily withstand intranasal challenge of at least a million lethal doses of highly virulent field strains of virus 7 to 10 days and 5, 7 and 10 weeks later. . . . In contrast, a negative or irregular immune response is obtained when the same or much greater quantity of the Blacksburg strain of virus is administered to passively immune birds via unnatural portals of entry.

The observations of Cox and his associates on the adaptation of MEF1 poliomyelitis virus to chick-embryos and its subsequent administration to humans are most encouraging and represent some measure of success following persistent and careful laboratory manipulation under carefully controlled conditions.

The virus of poliomyelitis was one of the few viruses that had resisted adaptation to growth in the tissues of developing chick-embryos. The adaptation of the MEF1 strain took four years in a laboratory that handled at one stage 14,000,000 fertile eggs *per annum* (Cox, 1953). It was started with the idea, not so much of developing a vaccine, as for producing a complement-fixing antigen for diagnostic work. Suckling hamsters were first used and 150 consecutive passages of infected suckling hamster brain given intracerebrally were made. This brought about a modification, which was evident from the fact that the virus content of suckling hamster brain eventually reached an I.D.₅₀ of $10^{-6.5}$ to $10^{-7.0}$ as determined by the usual intracerebral inoculation tests in adult mice. The 115th and 119th passages were established in chick embryos by the yolk-sac route of inoculation and the 131st hamster passage following allantoic inoculation. The virus could be maintained in chick embryos in its altered form, but the embryos did not appear to suffer an acute infection as they often do with encephalitis viruses, but died just prior to hatching. Virus was usually harvested five days after the inoculation of seven-day-old embryos. This suckling hamster-chick embryo adapted MEF1 virus still paralysed 10 of 70 monkeys after intracerebral inoculation. It was this virus that was recently fed to human volunteers without mishap (Koprowski *et alii*, 1952). Firstly, infected brains and cords of cotton rats were fed to 20 human volunteers. All remained asymptomatic, although most became carriers. Furthermore, all who did not have

antibody prior to ingestion of the virus developed detectable specific antibody after its ingestion. Later another experiment was carried out on 61 children (Koprowski *et al*, 1953). All remained asymptomatic; a carrier state developed in 29, and a specific antibody rise was demonstrated in nearly every case. Cox (1953) makes an interesting comparison of this MEF1 strain with the 17D chick-embryo-adapted yellow fever virus. He claims that 6% of monkeys inoculated intracerebrally with the 17D vaccine died from yellow fever encephalitis, while a further 15% developed slight weakness.

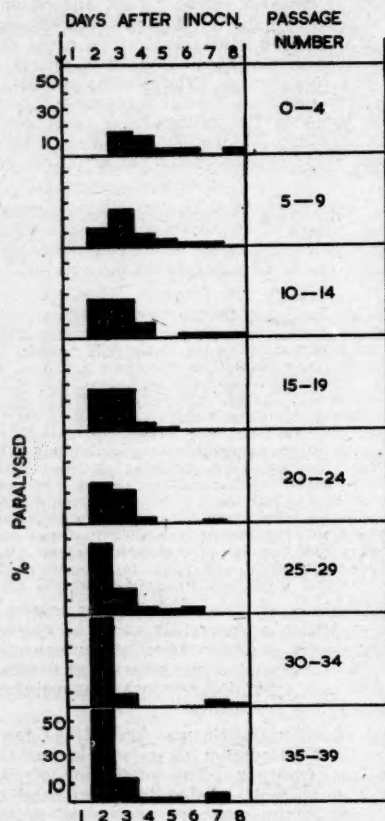


FIGURE I.
Percentage of mice developing paralysis after intracerebral inoculation with type I poliomyelitis virus during 40 consecutive passages.

It is our sincere hope that Cox succeeds in adapting both type I and type III poliomyelitis viruses to growth in the developing chick embryo, as they are the antigenic types commonly associated with severe epidemics and outbreaks. Unfortunately, they behave differently from the MEF1 type.

Sydney Research.

I am now in a position to present to you some of the experimental work we have recently been carrying out, as this has now been published (Stanley *et al*, 1954). Our first intentions were to develop a quick and accurate test for the estimation of poliomyelitis antibody in patients' serum and in pooled human serum as supplied by the Red Cross Blood Transfusion Service, Sydney, without recourse to tissue cultures. This was forced upon us by the fact that the pooled human serum contained "Merthiolate", which combined with a serum constituent to form a

complex which caused tissue degeneration (Stanley and Ponsford, 1954). When, therefore, it was observed by LI and his associates (1951, 1953) that both Mahoney (type I) and Leon (type III) strains of poliomyelitis could infect mice by the intraspinal route, we applied for and received these strains of virus. We are indeed grateful to the Alabama scientists, and to Dr. Schaeffer in particular, for providing us with their intraspinal adapted strains. Both types caused immediate paralysis and death when administered to our mice by the intracerebral route, but only rarely by the intraspinal route. Such an observation had not been made before, and I am of the opinion, until the results of further tests become available, that the genetic constitution of our mice is largely responsible for this

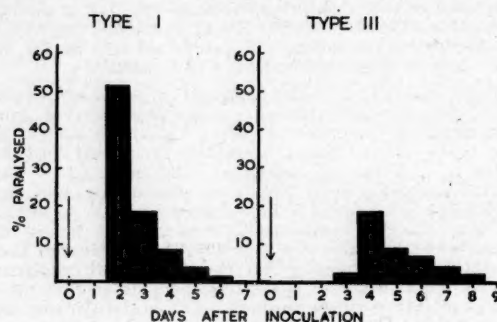


FIGURE II.
Percentage of mice paralysed after intracerebral inoculation with types I and III mouse-adapted poliomyelitis viruses.

extraordinary behaviour. This was fortunate, as it enabled us to select continuously virus which had an affinity for mouse central nervous system tissue. This was accomplished by giving virus intracerebrally and harvesting it from the cords of paralysed mice—in other words, selecting virus that was capable of invading mouse central nervous system tissue. During the course of 40 consecutive

TABLE I.
Neutralization Tests with Sera by the Use of Type I Mouse-adapted Poliomyelitis Virus by the Intracerebral Route.

Patients with a Clinical Diagnosis of	Code Number.	Time After Onset. (Days.)	Reciprocal of Titre (50% End-Point).
Paralytic poliomyelitis.	B339	7	240
		20	> 1000
	L333	32	> 1000
		40	> 1000
Encephalitis.	H710	5	40
		12	40

passages with type I virus and 20 passages with type III virus, some interesting observations have been made. The most important are as follows:

1. The following differences were noted between types I and III: (i) an increase with passage in the percentage of type I infected mice paralysed; this increase is not obvious with type III (see Figure I); (ii) a two to three day incubation period for type I and a four to seven day incubation period for type III (see Figure II); (iii) a greater percentage of mice paralysed with type I (see Figure II).

2. Almost all animals were paralysed in their forelimbs and not in their hind limbs. Of 2500 mice inoculated, 804

were paralysed in their forelimbs and 13 (1.6%) in their hind limbs. There were 49 (2%) non-specific deaths.

3. The virus was found in higher concentration in the cervico-thoracic area of the cord than in the lumbar area.

4. Both types failed to cause paralysis in monkeys when administered by the intracerebral route. Such monkeys were resistant to intracerebral challenge with the original paralytic strain.

5. The type-specific antibody prevents paralysis in mice, and the neutralization test is sensitive enough to titrate the antibody (see Table I). Further observations on the detection of neutralizing antibodies to type I mouse-adapted virus appear elsewhere (Stanley *et alii*, 1954).

There immediately appear to be two dangers, although I think they are slight, when one considers the possible use of a mouse-adapted living attenuated poliomyelitis virus vaccine (MALAP) for humans. Firstly, there is the possibility of contaminating the preparation with a latent virus from mouse-stock (for example, lymphocytic choriomeningitis or Theiler's encephalomyelitis virus). We have been attempting to isolate both these agents in Sydney for many years, but have not yet been successful. Secondly, there is the danger of a reversion to original virulence when the adapted virus comes into contact again with human tissue. Such, however, has not yet been the case with the other live virus vaccines mentioned earlier.

We think, then, that there is now considerable hope that the immunization of children with a live vaccine by the natural route of infection may soon be attempted. Should an effective vaccine soon become available, I sincerely hope, as Professor Cadham has suggested, "that it will not require as long to effect the eradication of poliomyelitis from our society as it has done and is doing for the elimination of smallpox following the discovery by Jenner of an efficient vaccine for the prevention of that disease".

References.

- ABRAMSON, H. L., and GERBER, H. (1918), "Active Immunity in Experimental Poliomyelitis", *J. Immunol.*, 3: 435.
- AYCOCK, W. L., and KAGAN, J. R. (1927), "Experimental Immunization in Poliomyelitis", *J. Immunol.*, 14: 85.
- BRODIE, M. (1935), "Active Immunization in Monkeys Against Poliomyelitis with Germicidally Inactivated Virus", *J. Immunol.*, 28: 1.
- BURNET, F. M. (1945), "Virus as Organism", Harvard University, Cambridge, Massachusetts.
- BURNET, F. M. (1951), "Some Biological Implications of Studies on Influenza Viruses: Ecological Approach to Common Virus Diseases of Today", *Bull. Johns Hopkins Hosp.*, 88: 57.
- CARASSO, V. J., and COX, H. R. (1949), "Propagation of Canine Distemper Virus on Chorio-allantoic Membrane of Embryonated Hen Eggs", *Proc. Soc. Exper. Biol. & Med.*, 71: 246.
- CLANCY, C. F., COX, H. R., and BOTTORFF, C. A. (1949), "Laboratory Experiments with Living Newcastle Disease Vaccine", *Poult. Sc.*, 28: 58.
- COWIE, D. M. (1935), "Protection of Monkeys Against Intracerebral Inoculation of Virulent Poliomyelitis Virus by Vaccination with Phenolized Poliomyelitis Vaccine", *Proc. Soc. Exper. Biol. & Med.*, 32: 632.
- COX, H. R. (1953), "Viral Vaccines and Human Welfare", *Lancet*, 2: 1.
- COX, H. R. (1953), "Active Immunization Against Poliomyelitis", *Bull. New York Acad. Med.*, 29: 943.
- DICK, G. W. A., SCHWEDT, C. E., HUBER, W., SHARPLESS, G. R., and HOWE, H. A. (1951), "Immunization of Cotton Rats with Inactivated Lansing Poliomyelitis Virus. II. Inactivation by Physical Methods", *Am. J. Hyg.*, 53: 131.
- ENDERS, J. F., WELLER, T. H., and ROBBINS, F. C. (1949), "Cultivation of the Lansing Strain of Poliomyelitis Virus in Cultures of Various Human Embryonic Tissues", *Science*, 109: 85.
- HABEL, K., and LI, C. P. (1951), "Intraspinal Inoculation of Mice in Experimental Poliomyelitis", *Proc. Soc. Exper. Biol. & Med.*, 76: 357.
- HAMMON, W. McD. (1953), "Passive Immunization Against Poliomyelitis", *Bull. New York Acad. Med.*, 29: 930.
- HAMMON, W. McD., SATHER, G. E., and HOLLINGER, N. (1950), "Preliminary Report on Epidemiological Studies on Poliomyelitis and Streptococcal Infections", *Am. J. Pub. Health*, 40: 293.
- HORSTMANN, D. M. (1953), "The Epidemiology and Pathogenesis of Poliomyelitis", *Bull. New York Acad. Med.*, 29: 910.
- HOWE, H. A. (1952), "Antibody Response of Chimpanzees and Human Beings to Formalin-Inactivated Trivalent Poliomyelitis Vaccine", *Am. J. Hyg.*, 56: 265.
- KASAHARA, M. (1939), "Über die Immunitätsverhältnisse des mit Ultraschallwellen behandelten Virus bei experimenteller Affenpoliomyelitis", *Klin. Wchnschr.*, 18: 971.
- KOLMER, J. A. (1938), "The Present Status of Methods for the Prophylaxis of Acute Anterior Poliomyelitis", *Ann. Int. Med.*, 12: 95.
- KOPROWSKI, H., and COX, H. R. (1948), "Occurrence of Rabies Virus in Blood of Developing Chick Embryo", *Proc. Soc. Exper. Biol. & Med.*, 68: 612.
- KOPROWSKI, H., JERVIS, G. A., and NORTON, T. W. (1952), "Immune Responses in Human Volunteers upon Oral Administration of a Rodent-Adapted Strain of Poliomyelitis Virus", *Am. J. Hyg.*, 55: 108.
- KOPROWSKI, H., JERVIS, G. A., NORTON, T. W., and NELSEN, D. J. (1953), "Further Studies on Oral Administration of Living Poliomyelitis Virus to Human Subjects", *Proc. Soc. Exper. Biol. & Med.*, 82: 277.
- LEVADITI, C., and LANDSTEINER, K. (1910), "Recherches sur la paralysie infantile expérimentale", *Compt. rend. Acad. d. sc.*, 150: 131.
- LI, C. P., and HABEL, K. (1951), "Adaptation of Leon Strain of Poliomyelitis to Mice", *Proc. Soc. Exper. Biol. & Med.*, 78: 233.
- LI, C. P., and SCHAEFFER, M. (1953), "Adaptation of Type I Poliomyelitis Virus to Mice", *Proc. Soc. Exper. Biol. & Med.*, 82: 477.
- LI, C. P., and SCHAEFFER, M. (1953), "Further Modification of the Mouse Adapted Type III Poliomyelitis Virus", *Proc. Soc. Exper. Biol. & Med.*, 83: 706.
- MARKHAM, F. S., BOTTORFF, C. A., and COX, H. R. (1951), "The Conjunctival Application of Newcastle Disease Vaccine (Intranasal Type) in Parentally Immune and Susceptible Chicks", *Cornell Vet.*, 41: 267.
- MELNICK, J. L. (1951), "Differences in the Degree of Infectiousness of Two Related Strains of Poliomyelitis Virus Following their Oral Administration to Monkeys", *J. Immunol.*, 67: 219.
- MELNICK, J. L., and LEDINKO, N. (1951), "Antibody Levels in a Normal Young Population During an Epidemic of Poliomyelitis", *Am. J. Hyg.*, 54: 354.
- MILZER, A., OPPENHEIMER, F., and LEVINSON, S. O. (1945), "A New Method for the Production of Potent Inactivated Vaccines with Ultraviolet Irradiation; Completely Inactivated Poliomyelitis Vaccine with Lansing Strain in Mice", *J. Immunol.*, 50: 331.
- MILZER, A., LEVINSON, S. O., SHAUGHNESSY, H. J., JANOTA, M., VANDERBOOM, K., and OPPENHEIMER, F. (1954), "Immunogenicity Studies in Human Subjects of Trivalent Tissue Culture Poliomyelitis Vaccine Inactivated by Ultraviolet Irradiation", *Am. J. Pub. Health*, 44: 26.
- MORGAN, I. M., HOWE, H. A., and BODIAN, D. (1947), "The Role of Antibody in Experimental Poliomyelitis; Production of Intracerebral Immunity in Monkeys by Vaccination", *Am. J. Hyg.*, 45: 379.
- MORGAN, I. M. (1948), "Immunization of Monkeys with Formalin-Inactivated Poliomyelitis Viruses", *Am. J. Hyg.*, 48: 394.
- MOYER, A. W., ACCORTI, C., and COX, H. R. (1952), "Poliomyelitis; Propagation of the MEF1 Strain of Poliomyelitis Virus in the Suckling Hamster", *Proc. Soc. Exper. Biol. & Med.*, 81: 513.
- PELTER, M., DURIEX, C., JONCHÈRE, H., and ARQUIE, E. (1940), "Vaccination mixte contre la fièvre jaune et la variole sur des populations indigènes du Sénégal", *Ann. Inst. Pasteur*, 65: 146.
- POLLARD, M. (1951), "The Inactivation of Poliomyelitis Virus by Freeze-Drying", *Texas Rep. Biol. & Med.*, 9: 749.
- ROBBINS, F. C., ENDERS, J. F., WELLER, T. H., and FLORENTINO, G. L. (1951), "Studies on the Cultivation of Poliomyelitis Viruses in Tissue Culture: the Direct Isolation and Serologic Identification of Virus Strains in Tissue Culture from Patients with Nonparalytic and Paralytic Poliomyelitis", *Am. J. Hyg.*, 54: 286.
- RHOADS, C. P. (1930), "Immunization with Aluminium Hydroxide Mixtures of Poliomyelitis Virus", *Science*, 72: 608.
- RHOADS, C. P. (1931), "Immunization with Mixtures of Poliomyelitis Virus and Aluminium Hydroxide", *J. Exper. Med.*, 53: 399.
- ROCA-GARCIA, M., MOYER, A. W., and COX, H. R. (1952), "Poliomyelitis; Propagation of MEF1 Strain of Poliomyelitis Virus in Developing Chick Embryo by Yolk Sac Inoculation", *Proc. Soc. Exper. Biol. & Med.*, 81: 519.
- SABIN, A. B. (1931), "Purification of Poliomyelitis Virus by Adsorption and Elution", *Proc. Soc. Exper. Biol. & Med.*, 29: 59.
- SABIN, A. B. (1951), "Paralytic Consequences of Poliomyelitis Infection in Different Parts of the World and in Different Population Groups", *Am. J. Pub. Health*, 41: 1215.

- SABIN, A. B. (1953), "Present Status and Future Possibilities of a Vaccine for the Control of Poliomyelitis", *Am. J. Dis. Child.*, 86: 301.
- SALE, J. E. (1953), "Studies in Human Subjects on Active Immunization Against Poliomyelitis; a Preliminary Report on Experiments in Progress", *J.A.M.A.*, 151: 1081.
- SALE, J. E. (1953), "Use of Adjuvants in Studies on Influenza Immunization; Degree of Persistence of Antibody in Human Subjects Two Years after Vaccination", *J.A.M.A.*, 151: 1169.
- SHAUGHNESSY, H. J., HARMON, P. H., and GORDON, F. B. (1930), "Heat Resistance of the Virus of Poliomyelitis", *J. Prev. Med.*, 4: 149.
- STANLEY, N. F., DORMAN, D. C., and PONSFORD, J. (1954), "Infection of Mice with Types I and III Poliomyelitis Virus Following Intracerebral Inoculation", *Proc. Soc. Exper. Biol. & Med.*, 85: 454.
- STANLEY, N. F., and PONSFORD, J. (1954), "Merthiolated Serum as a Cause of Fibroblastic Degeneration in Tissue Cultures", *Australian J. Sc.*, 16: 189.
- STANLEY, N. F., DORMAN, D. C., and PONSFORD, J. (1954), in the press.
- THEILER, M. (1952), in "Viral and Rickettsial Infections of Man", edited by Rivers, T. M., Philadelphia.
- THEILER, M., and SMITH, H. H. (1937), "Use of Yellow Fever Virus Modified by In Vitro Cultivation for Human Immunization", *J. Exper. Med.*, 65: 787.
- WELLS, T. H., ROBBINS, F. C., and ENDERS, J. F. (1949), "Cultivation of Poliomyelitis Virus in Cultures of Human Foreskin and Embryonic Tissues", *Proc. Soc. Exper. Biol. & Med.*, 72: 153.

AN APPROACH TO NEUROSES IN GENERAL PRACTICE.

By M. G. JANSEN,
Ororoo, South Australia.

IN common with Chapman (1953), I believe that the psycho-neuroses are sadly neglected in general practice. His article prompted me to record my experiences with them, in an endeavour to confirm that those commonly encountered in everyday practice are neither as difficult nor as hopeless as is generally considered, and that the time involved in their treatment is not excessive. The method of diagnosis and treatment which will be described produces gratifying results.

This article is presented, honestly and humbly, by a general practitioner with no post-graduate qualifications, as one approach to the problem of the common neuroses. A practical working plan evolved during seven years in one practice will be submitted. If it brings forth criticism, either from other general practitioners or from psychiatrists, this comment and discussion should help to clarify our ideas. My experiences are presented in the hope that they will stimulate interest, rather than add to the store of knowledge, in a young but virile branch of medicine. These experiences in psychiatry, which were born of necessity, led to a change in my attitude towards these so-called "nervous" patients, progressing from helplessness to increasing confidence.

A Changing Outlook Towards Neuroses and Psychotherapy.

Shortly after I graduated, during my period of twelve months in residence and subsequently as *locum tenens* in various practices for eighteen months, I had, in common with most of my colleagues, a rather hopeless and impotent attitude towards neurotic patients. Some were helped by simple reassurance; some were helped and others apparently made worse by the knowledge that they had no serious bodily disorder; some with current anxieties were cured by a simple explanation of the association between their symptoms and their anxieties and occasionally by removing or modifying the cause of their anxiety; but there remained a whole host of others who simply went on and on being a misery to themselves and to all around them. One could scarcely repress a shudder when they entered the surgery door. The general attitude towards them appeared to be one of apathy and resignation, and there was a tendency to regard them as nuisances, made bearable only by the

fact that they were a constant source of income. These neurotics appeared to be more prevalent in some practices than in others, but I could not decide whether this was because some doctors would, by their handling of patients, breed neuroses, or because they were able to give their patients a little more comfort and moral support, and so collected them. Certainly some doctors could relieve anxiety and help the distressed to bear their burdens more cheerfully without prescribing a bottle of mixture.

When about two and a half years after graduation I commenced practice at Ororoo, as the sole medical practitioner in a small country town 170 miles north of Adelaide, with a population of 1500 to 2000 in my district, I had no clear idea of how I was to deal with neurotics. Our teaching in psychiatry had been meagre. I thought I could distinguish between a psychosis and a neurosis, and between a neurosis and a bodily illness—but, alas, how wrong I was!

I well remember one of my lecturers basing his teachings on the writings of T. A. Ross, and another eminent honorary stating that he endeavoured to read "The Common Neuroses" by Ross once in every year, and exhorting his students to do the same. I had read and reread this delightful book, and intended to follow his teachings as much as time and circumstances would permit; but I was not particularly optimistic, as I could find no clear example to follow among my colleagues. I also read—or rather attempted to read—other books on psychiatry and neuroses, but found them either too technical or otherwise so much confused by theoretical considerations as to be almost unintelligible to my limited comprehension.

Shortly after arrival at Ororoo I was confronted with several young adults with hysterical manifestations, and these were treated by sedation, followed later by several interviews of one hour's duration, in which their life histories were taken, their anxieties or conflicts determined, and the mechanism of production of their symptoms explained. These results were encouraging, but all my resolutions suffered, and despair resulted, from contact with my quota of those chronic neurotics who had a long list of bizarre symptoms and remarkably healthy bodies often carrying multiple operative scars. Their convictions and prejudices seemed too strong to be readily influenced; suggestion and reassurance seemed valueless; to invite them back for longer interviews seemed too horrifying—and anyway unlikely to be useful—especially when one's spare time and leisure were limited. Rather by accident than design, however, an opportunity sometimes presented itself to show these patients, to their satisfaction, how anxiety had aggravated their symptoms. This would produce some relief and provide a useful stepping-stone to further simple psycho-therapeutic measures and increased relief. It became worthwhile assiduously to seek this opportunity, rather than to attempt reassurance alone. Occasionally, obstinate refusal to provide a prescription, accompanied by repeated assertion that their symptoms were due to emotional causes, illustrated by simple examples, would ultimately produce relief, although it seemed more commonly to drive the patient away to other more accommodating physicians. Mostly it seemed easiest to listen to them for a while, examine them if necessary, and endeavour to give them some medicinal relief. Attempts to induce these patients to consult a psychiatrist were generally fruitless, either because they could not afford the time and money or because they would not believe that it would be useful to them. In general, psychotherapy seemed well worth attempting, but there were many practical considerations, principally the time factor and lack of knowledge or ability, which often forbade its application.

Two years ago my interest in psychiatry was awakened for two reasons. The first was an increasing recognition of the fact that some patient's progress over the years had disproved my original diagnosis, which, fortunately for my peace of mind, had usually been supported by consultants. The commonest error had been to miss the diagnosis of a neurosis. The second was the presence of several patients with chronic neuroses whose mental and physical conditions were deteriorating and whom I did not feel competent to treat. These patients genuinely could

not, for domestic or financial reasons, conveniently undergo prolonged treatment by a psychiatrist. Reassurance, guidance and supportive therapy were no longer helpful to them.

General practice in the country gives one unique opportunities to observe the patient's progress and his reaction to environmental influences and to obtain his opinion and that of his friends and intimate associates. It also makes it well-nigh impossible to overlook or evade one's mistakes and responsibilities.

After much consideration and hesitation I decided to attempt to treat these patients by simple analytical therapy. By comparison with similar reported cases, I expected this to take many interviews, but to my surprise these patients were relieved or cured within a few weeks. I also sought a better method of history-taking and examination, applicable to the circumstances of general practice, in order to minimize the errors of diagnosis. The immediate results of these two efforts were excellent, but several personal doubts and fears remained: (i) The patient, his friends and I were well satisfied with the result; but would it be permanent? I had previously seen two patients show temporary improvement, but later retrogression. (ii) Treatment of these conditions had been too easy, too good to be true and not in conformity with what I had read. I had been looking for major guilt complexes and repressed sexual causations as predisposing causes, but commonly found feelings of frustration, insecurity or, as Hadfield (1952) postulates, a feeling of deprivation of love. Often the patient got better before I was ready to form an interpretation of the psychopathology of his illness. It was even better than Ross's teachings led one to believe; but this was at the same time disconcerting. (iii) Contact with my fellow practitioners was limited, but among these I could find no echo of my own experiences. I was then unorthodox.

These doubts and fears gradually disappeared. The patients remained well for a sufficient period of time to be confident of the long-range results. Chapman's article (1953) then showed me that at least one other colleague was working along similar lines and getting similar results in general practice. An excellent book by Watts and Watts on "Psychiatry in General Practice" (1952) has been a practical, comprehensive and convincing guide to this work. "Counselling and Psychotherapy" by Rogers (1952) has enabled me to improve and shorten my technique of treatment. "Psychology and Mental Health" by Hadfield (1952) has produced a much clearer conception of the sources of behaviour and the origin of personality traits and neuroses.

More recently I have attempted to treat by psychotherapy some diseases of doubtful etiology which have failed to respond to orthodox physical therapy. Again, the results have been gratifying. These cases have, however, been selected, the numbers are small, and any generalizations at this stage would be dangerous.

The following case illustrates how the diagnosis of neurosis was originally missed and how structural changes developed, and one method of applying treatment.

CASE I.—A young man, aged twenty-two years, consulted me in 1949, complaining of diarrhoea of two months' duration, which was steadily becoming worse. He had from three to seven watery bowel actions per day. He had had occasional intermittent diarrhoea since the age of fifteen years. He said that he was easily excitable, and that excitement would aggravate the diarrhoea. Examination of the patient revealed indefinite tenderness and possible spasm of the descending colon. His symptoms became worse despite reassurance and medicinal therapy. X-ray examination with a barium enema revealed narrowing of his colon and decreased haustration with rapid passage of the barium around the colon. Sigmoidoscopic examination revealed a red granular mucosa, bleeding easily on contact. These findings appeared to make the diagnosis of neurosis untenable. More intensive therapy limited his motions to three or four per day, but an exacerbation of symptoms occurred every few months. He began to limit his diet increasingly and became more concerned. He was referred to an eminent consultant, who confirmed the diagnosis of colitis and prescribed further medicines, including opium, and dietary restrictions. His condition remained unaltered.

In 1953, when he reported for a routine review of his condition, I explained to him how it was becoming increasingly recognized that repressed emotion was a common causative factor in colitis. I suggested that a full investigation of his emotional state should be undertaken, and told him how this was done. Immediately and with considerable emotion he told me that he could not easily talk of his mother's death, which had occurred when he was five years old. An interview out of ordinary hours was arranged, his life history taken, and self-expression encouraged. This disclosed that he had retained his infantile attitude towards his deceased mother, and that his symptoms had commenced at the age of fifteen years, when his father had remarried. His mental attitude towards this marriage was determined. Several interviews were spent in helping him to gain more complete insight. His symptoms steadily disappeared.

Several minutes spent on a well-conducted inquiry into his emotions at his first interview would have saved him from four years of ill health.

Incidence of Neurosis in General Practice.

Most practitioners will readily agree that neuroses comprise a considerable proportion of the cases seen in general practice, and after reading Chapman's article I was prompted to determine their percentage in mine.

I first attempted to classify cases into either bodily or neurotic disorders, but there was a substantial proportion of cases in which such a classification was difficult, such as the following.

CASE II.—A middle-aged woman presented with precordial pain, commencing without apparent cause. She had had a similar pain six weeks before, following a muscle strain. Further questioning revealed that she had felt tired for two months and that there had been slightly increased pain and bleeding during her last two menstrual periods. Her husband had died three years before of congestive cardiac failure. I knew her to be a hard-working, well-adjusted woman who, having two children to support, had a difficult life, but who accepted this burden cheerfully and competently. The only abnormality revealed on examination was an enlarged, irregular uterus containing fibroid tumours. During the subsequent discussion the patient hesitantly revealed that she was afraid she was developing heart trouble; she had nursed her husband for several years, and the slight debility and precordial pain had resembled his symptoms and produced her fear. She had not associated her debility and pain with her menstrual changes. She readily recognized the sequence and association of events when her fear was expressed.

This case could not be regarded as a pure neurosis; but neither could the patient's symptoms be explained without consideration of the psychic influences. Moreover, treatment of her fibroid tumours would not cure her pains if the emotional influence became more firmly fixed or repressed.

The following three groups of nervous diseases may be differentiated: Group A: neuroses, in which the causation is emotional, and in which symptoms are relieved after these influences cease or are dealt with; Group B: proven psychosomatic diseases, in which there is a somatic abnormality the causation of which is predominantly emotional, and in which symptoms and signs are relieved with psychotherapy, as in Case I; Group C: unproven psychosomatic diseases and emotional reinforcement of organic disease, in which emotional influences aggravate or complicate a somatic disorder, but in which they cannot be shown to be the predominant cause, as in Case II. Many of these would probably be included in Group B if a more complete investigation of the psyche was attempted. Other patients may later develop a neurosis if the psychic aspect of their disorder is neglected.

The numbers and percentages in these groups, determined in 1204 consecutive medical attendances on 463 patients during three months, are given in Table I.

This incidence of neuroses is higher than that determined by Chapman (8%) and those determined in England. Three reasons may be given for this, all of which have been recognized by Chapman: (i) As I have been in this practice for seven years, the character and life history of these patients are known. This materially aids diagnosis. (ii) The patients seen by a *locum tenens* are not necessarily representative of those seen by the principal.

(iii) The time available per patient here is greater than in England, and this affects the accuracy of diagnosis. I had determined the percentage of cases of neurosis during a busier three-month period before, and found it to be 10%.

TABLE I.

Group.	Proportion of Total (1204) Attendances.	Proportion of Total (463) Patients.
Group A ..	147 (12.2%)	59 (12.7%)
Group B ..	61 (5.1%)	25 (5.4%)
Group C ..	179 (14.8%)	86 (18.6%)

Diagnosis.

Ross (1942), Thorner (1948) and other authors all stress that the diagnosis of a neurosis should not be made by the absence or exclusion of metabolic or anatomical disease alone, but that it is an independent one. It has its own positive as well as negative features, and can be made whether other disease exists concurrently or not. This bears reiteration; we should all know it, but we all forget or overlook it. These positive features are well described by Ross, and are as follows: (i) the bizarre and often contradictory nature of the symptoms; (ii) the presence of other nervous manifestations; (iii) the attitude of the patient towards his symptoms; (iv) the whole life history.

A reasonably adequate history and examination will reveal some or all of these features in a neurotic patient, so long as one is alert and looking for them. In a few patients the diagnosis is clearly defined at the first interview; but there is a substantial proportion in whom the findings are inconclusive. It remains uncertain how much of the patient's complaints rests upon anatomical or metabolic change, and how much may be of emotional origin.

The problem sometimes seems insuperable for several reasons, the principal ones being as follows: (i) The symptoms may be trivial or vague, and could be either those of an anxious person or those of a person with some organic disease which shows little or no bodily signs. (ii) There may be multiple complaints and bodily abnormalities. (iii) There may be inconsistencies between the nature of the complaint and the findings on examination. (iv) The clinical picture presented may have an uncertain or varied aetiology.

Under these conditions the general tendency is to attempt to exclude organic disease, or assess its severity, by further investigation. This has the virtue of avoiding the criticism of our patients and colleagues, should we miss the diagnosis of a serious disease like tuberculosis or carcinoma; but it also has the disadvantage of more commonly getting negative results, wasting time and money, and still failing to make a definite and positive diagnosis. Experience has shown that this method also frequently breeds or aggravates a neurosis.

In view of the prevalence of neuroses and anxiety symptoms, it is logical that the patient's emotional system should be investigated at least as thoroughly as any other bodily system. This can be time-consuming, especially when the life history is examined in detail. I have tried many variations and combinations of history-taking and examination in an endeavour to make an accurate diagnosis with a minimum of time expenditure. The most valuable and conclusive method has been to take the history, examine the patient, and then investigate his emotional attitude towards his symptoms.

This approach to diagnosis must be amplified a little, as some seemingly trivial details are important.

The history is taken by allowing the patient to relate his complaints and then asking pertinent questions about these, the past history, family history and the function of the other bodily systems, being ever alert to observe the apparent contradictions or inconsistencies and the nervous manifestations which distinguish a neurosis. Rarely, the

patient will spontaneously relate or ventilate his anxieties. If this occurs it is best allowed to continue, so long as time permits, when it may be terminated as follows: "I cannot give you any more time now, as you will understand. These anxieties may have been the cause of your troubles, and releasing them may relieve or help you, but will at any rate produce a better understanding of your illness." The patient can be examined at the next consultation.

The examination in general practice is often incomplete, and rightly so. If the patient simply has a cut finger, or wax in his ears, he will generally be resentful if you start prodding his abdomen; but when the diagnosis is not clear examination must be complete and thorough. This is necessary even when a "spot" diagnosis of neurosis can be made, since this may have been caused or aggravated by a physical illness, as in Case II. Also, the anxious person may contract a physical illness.

During, or on completion of, the physical examination the patient usually asks what you have found or what is wrong with him. This question I answer truthfully: "I can find no evidence of any bodily troubles"; or "There is some tenderness, the causation of which is uncertain". The effect is always that the diagnosis remains uncertain and that further investigation is needed. No suggestion that it is due to emotional causes should be made, as this often arouses resentment. The inquiry usually follows smoothly from the patient's questions. If an opening gambit is necessary, it is almost as awkward and unsatisfactory as the first question put to the patient as he enters the surgery. "What do you think your trouble is due to?" is better than "Have you any fears or anxieties?" or "What are you worried about?" The last two questions imply suggestion, and while this may have some value by demonstrating your astuteness, it may also hamper the investigation. It is better to say: "I cannot be certain of the cause of your illness. It may be a disease in its early stages, or one which does not show any obvious signs. Quite commonly complaints like yours result from fears and anxieties. Could these be playing any part in your condition?" This is still slightly suggestive, but also indicates that you are receptive and ready to explore all possibilities.

We are now seeking not the symptoms, but the emotional relation and reaction to them; we are searching for the mental feeling aspects of the situation, and moreover, at this stage the immediate situation rather than the patient's past. Past history may become important later, but for diagnosis and therapy to take place it is by no means always necessary, and strangely, when there is no probing for the facts of the past history, a better picture of the dynamic development of the neurosis often emerges (Rogers, 1942).

Subsequent questions or remarks are not directive, but designed to create the impression that you wish to understand the patient's statements clearly and help him in self-expression. Speech is the principal link connecting the patient's mind and yours, and the principal means of communicating detailed information; but its value may be jeopardized at either end of that linkage by poor ability to present one's ideas, or by difficulty in interpreting that information. We must determine the patient's actual emotional feeling and not interpret his statements by comparison with our own personal or professional experience. To learn, for instance, that the patient's wife has deserted him is not sufficient—he may be happy to be rid of her.

The following two examples are typical of the application of this method.

CASE III.—A woman, aged seventy years, had had upper abdominal discomfort and vomiting, commencing three days before seeking medical advice, and continuing for thirty-six hours. Since then she had felt nauseated. She had been active before this illness, but no longer felt able to get out of bed. She had had similar milder episodes during the previous twelve months, and frequent "rheumatic" pains. She was well preserved for her age, had an anxious and flushed facies, and was afebrile. The significant abnormalities were an enlarged heart, a blood pressure of 210

millimetres of mercury, systolic, and 110 millimetres, diastolic, and slight tenderness over her gall-bladder. Inquiry into her emotional state revealed that she always got dyspepsia with anxiety. She had been disturbed shortly before her symptoms began by a fire involving one of her properties. During the previous eighteen months there had been many difficulties and anxieties associated with the estate of her late husband, who had died suddenly after a coronary occlusion. Her parents, brothers and sisters had all died at or before the age of seventy years, of hypertension or cerebral vascular accidents. Her mother had had many attacks of dyspepsia like hers. She did not know what would happen to her feeble-minded daughter if she died. One of her brothers had vomited his dentures shortly before he died; she had vomited hers three days earlier. There was abreaction during the relation of some of these episodes.

The whole consultation lasted forty minutes. The fifteen minutes spent on inquiry into her emotional state did establish that the cause of her present illness was emotional, although there were somatic abnormalities.

CASE IV.—A single woman, aged twenty-two years, complained of menstrual irregularity and pain. She was a healthy-looking girl and had no significant pelvic or general abnormalities. On being told this, and that she had a well-recognized condition, the causation of which was variable, she asked if her condition could be due to "nerves", and then related how she was generally healthy and happy, but disturbed by her attitude to sexual matters, men and marriage. She had little knowledge of sex and was afraid of it. She had never been told about menstruation, but had been put to bed during her first menstrual periods with no comment or explanation. The pain had begun some months later. In recent months she had remained active during menstruation, but was not sure whether that was right. She had never felt able to discuss her problems with her parents or anyone else. This consultation lasted thirty minutes.

So long as one adopts an attitude of sympathetic understanding and encourages free expression, this ventilation, or catharsis, and an abreaction seem to come freely. A similar inquiry before examination, or at a subsequent consultation after further investigation, is not nearly so effective in providing a maximum of diagnostic data in a minimum of time. It seems that the mental conditions are all "ripe" and ready for ventilation at this stage, and require only a little encouragement from the physician.

Before I adopted my present approach there were two aspects in the behaviour of patients, which I commonly observed, and which puzzled and distressed me. These were, firstly, hesitancy by the patient to end the interview and, secondly, a "perverse" response by him to a logical suggested course of treatment or investigation. These are both illustrated by the following patient.

CASE V.—A young man presented with symptoms resembling "nervous dyspepsia". Since there was a family history of peptic ulcer, and in order to reassure him, I advised him to have a barium meal X-ray examination. His objection seemed unreasonable, but further persuasion and explanation were of no avail, so I prescribed dietary measures and a powder as a trial for a few days. He was still hesitant to accept this and apparently did not want to terminate the interview. Subsequently he consulted at least three other doctors, with a repetition of the same advice and response. Finally he had the X-ray examination with negative findings.

This perverseness to reasonable and logical suggestions, and hesitancy to leave the surgery, have been reliable signs of a neurosis. Patients in whom it had been observed nearly always presented at a future time with a frank neurosis. This young man had a lot that he wanted to tell his physician, but he was not given the opportunity. The perverseness was not intentional or malicious, but simply an expression of his vague "subconscious" sense of dissatisfaction and disappointment.

The advantages of this general approach at the first interview, when the diagnosis is doubtful, are many. (i) It is reliable and minimizes mistakes. We can with assurance determine whether emotional influences are prominent in the causation of symptoms, and confidently proceed with treatment. (ii) It is relatively rapid and compatible with the conditions of general practice. (iii) It is effective whether the neurosis is acute or chronic. (iv) It requires little specialized knowledge of psychiatry. (v) It leads to a positive diagnosis. (vi) Other investigations can be performed subsequently, if necessary, without adding fuel to the fire of the patient's fears and

anxieties. He will have consciously recognized that emotion is playing some part in the causation of his symptoms. (vii) It avoids breeding neuroses. This happens easily. In investigating neurosis it is remarkable how often a well-intentioned and sometimes correct, but misinterpreted, remark by a medical practitioner (often myself) has played a significant part in causing, prolonging or perpetuating neurotic symptoms.

The diagnosis of the type of neurosis, or of the neurosis with psychotic tendencies, may be difficult, as few of these patients fit into any typical text-book picture. Several interviews may be required to make these distinctions, but they are generally of lesser consequence when one is in doubt, as they are determined during simple psychotherapy, which is often equally beneficial to the mildly psychotic patient.

Treatment.

There are many methods and techniques of psychotherapy, but the first essential in any is the encouragement of free expression. The success or failure of this encouragement depends principally on the attitude of the therapist—not only his words, but his whole demeanour. This attitude must be as natural as possible, since neurotics possess a remarkably shrewd intuitive insight into the character of others; but it must also be one of moral detachment and sympathetic understanding, since free expression will not continue in an atmosphere of censure or criticism. This attitude is difficult to assume at first, but becomes more natural as one learns to see not only the patient as he is, but also his latent or repressed potentialities. As Hadfield. (1952) states, the patient is cured by the faith of his physician in him.

Every physician must develop his own technique by trial and error, adjusting it as his experience and idiosyncrasy dictate. The developments in my own procedure, as practised at present, are here outlined. Acknowledgement must be made of my deep indebtedness to Rogers's description of his non-directive therapy, which has recently been of great value in confirming, clarifying and improving my own work.

The method of diagnosis by determining the emotional feeling content of the patient's symptoms has laid the foundation for therapy. The self-expression is allowed to continue and is encouraged. If the patient seems to have more to tell, the interview may be terminated, as indicated under "diagnosis", and an appointment made, preferably for some time out of ordinary consulting hours. However, it is worth spending a few extra minutes when ventilation is free, as, if he is interrupted, it may be difficult for the patient to resume later.

First Therapeutic Interview.

When, as in approximately one-third of the cases, ventilation appears to be complete, the patient's statements are clarified (Rogers). The patient may have described some disturbing situation, but his own emotional feeling reaction to it must be determined. By seeking this clear conception in your mind, you simultaneously create it in his. As in the above-mentioned example of the man deserted by his wife, his response may be one of pleasure and not pain, or he may be unhappy and ill at ease, not because his home has been broken up, but because his wife has also taken his pet dog, or because of guilt at his own feeling of relief. However, if the patient's story fits his symptoms an explanation may be given to him, such as the following: "Emotion can cause bodily disturbance, which is real and not imaginary [tears or blushes being used as simple examples]. This bodily disturbance may be prolonged after the emotion apparently ceases, by transference of interest (usually anxiety about symptoms), conditioned reflexes, or the (unconscious) advantage gained from the symptoms. This can be related to your anxieties and symptoms. The release of your emotions and the understanding you have gained may be sufficient to cure you. If you are not completely cured let me see you again."

The interpretation is frequently right, but is of no value to the patient until he is aware of it, understands and believes it. There is often resistance to this, as it may be

unpleasant. The interpretation, too, may be quite wrong; the more psychiatric work one does, the more one realises how little one knows about the patient, even after many interviews, and how often early impressions are disproved. To present strongly a wrong impression, or one that is right but not understood, will only confuse, disappoint or antagonize the patient, and later psychotherapeutic efforts will be made more difficult, or even impossible. A tentative explanation is, however, justified, so long as the patient feels free to return if necessary, and understands it to be provisional; for when his self-expression or ventilation has been good, it is often sufficiently correct to enable him to gain understanding and relief.

Actually, in these instances, little explanation is needed, as by the methods used the patient's insight grows almost as rapidly as the doctor's interpretation. In these cases there is little repression; the faulty associations may be either conscious or unwitting (Ross, 1942). The patient's reaction after explanation is akin to ours when a consultant gives us a diagnosis which we should have known ourselves, but did not think about because our thoughts had been confused by irrelevant issues. It is only to counter the mild feeling of shame or remorse that any explanation is needed.

Second Therapeutic Interview.

The second interview is often arranged to permit it to be of one hour's duration; but subsequent interviews are conducted during consulting hours and are limited to twenty or thirty minutes, according to the number of patients waiting. What is required is not so much time alone as an unhurried method of approach, which comes when one has confidence in what one is doing. Short interviews are desirable for the elderly or unintelligent. To allow the patient to come when he feels like it usually results in his coming when his emotional tension is heightened and conditions favouring emotional release are most opportune. The average spacing is one week.

The physical examination is first completed, if this has not already been done, and the findings are discussed. Self-expression and the limitations of speech are also briefly discussed, so that the patient understands the purpose of your statements or questions. A simple explanation of the mechanism of production of neuroses may be given. The patient's action in seeking assistance is recognized as the first step in his cure. The subsequent programme is defined; you are there to help him towards gaining insight and independence and, if necessary, to support him morally or physically during this process. This aid is necessary but temporary, comparable with the necessary support given by a splint or plaster while the bone heals. The common secondary fears of anxious patients—for example, those of insanity, suicide or disease—are often recognized and discussed at this stage. It is sometimes advisable to forewarn him that treatment may temporarily aggravate his tension. These explanations are usually given as a clarification or amplification of his own remarks, and only when it seems necessary to strengthen the feeling that he is understood, that there is a reason for his illness, and that his illness can be cured by his cooperation.

Progressive Stages of Free Expression and Clarification.

The pattern of subsequent proceedings is much the same in any interview and for any patient. The essentials are encouragement of free expression and emotional release, with remarks or questions designed only to clarify the feeling content. This may be difficult at first, especially when one is endeavouring to recognize and echo the expression of complex and ambivalent feelings. If this is correctly done, you will determine his true feelings and he will become fully aware of them, perhaps for the first time. His first expressions are usually obviously wrong, obviously "negative" (Rogers, 1942), and obviously a conscious cover for his subconscious frustration, inadequacy, fear or guilt. When these have been fully expressed and clarified, the positive or repressed feelings follow,

weakly and timidly at first, but growing in volume. There must be no endeavour to anticipate his feelings, though it is often difficult to suppress one's interpretation. There must be no endeavour to lead or guide the patient's thoughts or expressions in any direction. Inevitably they will lead through the precipitating emotional disturbance to the initial conflict or repression. This may take only a few minutes, when, as in Case II, the neurosis was on the conscious level; or it may take many interviews in stages, as Rogers well describes, when the conflicts are more deeply repressed. The time taken probably also depends on the patient's intelligence and character.

The patient must not be interrupted. His mental excursions may be into seemingly irrelevant issues, or something which you already know. It has surprised me to observe so frequently that the patient's story progresses through a series of disturbing events, which he has presented to me in the same order on previous occasions before serious psychotherapy was attempted. What I had failed to assess then was his particular emotional response to those events.

Clarification, correctly done, encourages further self-expression, progressing to an accurate knowledge of the psycho-dynamics of his neurosis. The patient sees more and more of himself and ultimately the completed picture, including the purpose served by the neurosis. It is unnecessary to present the interpretation to him—often one of the most difficult steps when other analytical methods are adopted. Positive, healthy impulses and actions begin without encouragement.

This spontaneous mental development, following effective and complete emotional release, and involving reassociation, reconditioning and sublimation, is fascinating to watch. It requires neither reassurance nor advice.

The patient may ask many questions. Few of these require an answer; it is more important to clarify the doubt or ambivalent feeling which these express. Consciously the patient may be seeking reassurance and advice, but unconsciously he is testing his and your belief in his latent potentialities. Advice will then only hinder the nurture of this belief.

Difficult and Unsuitable Cases.

The foregoing outline of treatment describes its course in the common cases, comprising about two-thirds of the total. In the majority of the remainder it soon becomes evident that environmental or social conditions are too harsh or hopeless, or that the patient is too old, too young, too dull or too unstable, or that he is deriving too much advantage from his symptoms to respond to non-directive therapy. In these cases limited insight can be given, but it may be essential, or at least easier and more efficient, to employ in addition environmental treatment, persuasion, suggestion or reassurance. There are a few neuroses in patients of extreme age or of poor constitutional make-up approaching psychopathy, in which little can be offered beyond sedation and the comfort given by one who understands.

If during the early interviews it is found that the apparent anxiety state is in reality an obsessive neurosis or a deep-seated phobia, or that there are definite psychotic features, then it is best to refer the patient to a psychiatrist. These interviews will have been useful in giving him a little insight and hope, and some conception of psychiatric methods, and in making him a little less afraid of the psychiatrist and what he implies. The manner in which he is referred to a psychiatrist may materially affect his prognosis (Kennedy, 1953).

Other Methods of Psychotherapy.

The starting point in the treatment outlined is the symptoms as they affect the patient. The best method of securing his cooperation is to deal with these, since he wants them cured. As Hadfield (1952) puts it:

The symptom is the royal road to the deep-seated causes of the disorder; the epitome of the disorder in the personality . . . It is the end product of more deep-seated disturbances, the resultant of the underlying complexes which beset the personality, the manifestation

in conscious life of unconscious disturbances, and, like the bubbles on the surface of the ocean, it directs us to the wreckage below.

Hadfield utilizes free association, whereas in Rogers's non-directive therapy free expression and clarification are used. The latter method is adequate, more rapid and more easily applicable for the common neuroses encountered in general practice.

Many other analytical methods may be employed. Watts and Watts (1952) describe those most suitable for general practitioners. They may need to be used, especially when the tension and stress created by the patient's symptoms are less than that involved in expressing his feelings about his problems. The method outlined above appears to produce results more quickly, does not require long interviews or copious notes, and avoids the difficulties associated with forming and presenting an interpretation. The patient's insight will develop only as rapidly as his character and intelligence permit.

Environmental treatment is most commonly employed after the patient has gained some insight, and as an additional aid to therapy. Often it is decided by the patient himself. It may be the sole treatment, as in teaching the mother how to handle her "nervous child" or as in helping those of poor character.

Suggestion, persuasion and reassurance are commonly used in gaining the patient's cooperation and in reinforcing the desire for cure. They can, when skillfully applied after rapport has been established, and in conjunction with a knowledge of psychopathology, produce a rapid cure by shattering a false belief or breaking a vicious circle. They are of great value in comforting and guiding a chronically affected patient, but their effect is usually only temporary.

Intellectual or moralistic advice may be well-intentioned and may produce temporary benefit; but generally it is futile and often harmful. It makes too severe a demand upon the patient, who is usually doing his best anyway.

Medical treatment such as sedation or measures to relieve secondary anaemia may be used if necessary, so long as the patient clearly understands the purpose in providing temporary relief. Bodily abnormalities may be treated simultaneously, and should be if they play a part in the causation of symptoms; but to attempt to cure a neurotic patient simply by putting him into first-class physical condition is nearly always futile. The patient's physical health may improve, but he feels no better.

The selection of the type of psychotherapy to be employed for any individual patient depends not only on his history, age, constitution and environment, but also on the personality, beliefs and attitude of his physician. It took me a long time to become clearly aware of the importance of this last-mentioned factor, which is ably described in Balint's (1954) article "Training General Practitioners in Psychotherapy".

Therapeutic Aims and Results.

The aim of therapy is not only to cure the symptom, but also the underlying symptom-complex, in order to enable the patient to attain healthy, self-directed action. Symptomatic cure may be achieved by many means, and sometimes more easily and quickly by medical treatment and reassurance; but a careful study of the life history of neurotic patients shows that this "cure" is only temporary. The symptom-complex manifests itself again, sometimes in another symptom or in a psychosomatic disorder. The ideal aim is to give the patient complete insight into all his problems, full and harmonious functioning of the whole personality or, in other words, complete mental health.

The assessment of results is not always easy. How are we to measure mental health? The symptom is an index of cure, for it disappears when the basic personality problem is solved; but it cannot be used alone, since the symptom may be cured without the basic problem being solved. The tabulation below is based not only upon relief from somatic and psychic distress, but upon the ability to attain healthy self-directed action and upon

social adaptation. The results given are impressions rather than statistics; but I do claim that they are reasonably accurate, as the medical and social progress of neurotics has been observed with more than ordinary interest, and opportunities for such observation are unique in a small country town.

In over two-thirds of neurotic patients symptomatic cure, psychological integrity and independence, and satisfactory social adaptation may be achieved. About half of these require one interview of half an hour; most of the remainder need from two to four interviews, and a few need more. These are mainly those with acute neuroses or acute exacerbations of chronic neuroses. Some of these results approach the ideal aim. In others, character traits which we may consider undesirable remain, but these are compatible with the patient's personality and daily experience.

In one-third of the remainder (or 10% of the total) there occur symptomatic relief and limited insight. These are mainly patients with chronic neuroses or patients of advancing age. The following is a good end result in this group.

CASE VI.—A married woman, aged thirty years, had become increasingly miserable since the age of sixteen years. Any minor illness would make her depressed and put her to bed for weeks. After three interviews of one hour, improvement commenced. After three more sessions her whole demeanour and outlook improved remarkably. I saw her twelve months later, during an epidemic of severe respiratory infections. She was no more miserable and responded as quickly as did the average patient. On inquiry into the result of psychotherapy her husband's response was most enthusiastic: "She is a new woman." (Perhaps he was over-enthusiastic because her frigidity had also been cured.) Her own response was: "I feel a lot better. Sometimes I get a bit miserable, and think I will come and have a few more talks, but within a day or two I get over it. I am well satisfied."

There is another 10% of the total which includes those for whom specialist attention is advisable, and others to whom the comfort given by sympathetic understanding, reassurance and medicines seems the only feasible therapy.

The remaining 10% of cases are either failures or errors. A rare source of error is the failure to detect a psychosis or a bodily disease. But the common source of failure, occurring in both acute and chronic cases, is psychological incompatibility. Some patients are frightened away to other practitioners; a few subsequently return when they learn by bitter experience that physical therapy or an operation has been useless. Most simply refuse or cease treatment. It is easy to blame the patient, attributing it to his obstinacy or "cussedness"; but the logical and real cause can be only my own blind spots, shortcomings and mistakes. The failures will be reduced only when I become clearly aware of these and amend them, in the same way that other failings have been corrected in the past.

Advantages of Treatment in General Practice.

There are several encouragements to treat neuroses in general practice. There is, firstly, their frequency (8% to 12% of patients, with another 20% or more in whom psychic factors are prominent in the symptomatology) and the mass of chronic suffering which they represent.

Secondly, a general practitioner has many advantages over a psychiatrist, since he sees the patients earlier, he has their confidence and knows their background, and he can commence therapy when the emotions are "ripe" for release. There is another big advantage, in that the practitioner can treat these patients without significantly altering their daily routine of living or their occupations, thus facilitating recovery as well as avoiding excessive inconvenience.

Thirdly, there is the fact that many neuroses, estimated by Watts and Watts (1953) at 25%, are either self-limiting or spontaneously cured by fortuitous environmental change or suggestion. Observation of such cases made me realize that effective treatment, although seemingly difficult at that time, was possible, and that it was worth seeking, and dealing with or removing, the predisposing cause.

We plead the disadvantage of lack of time and knowledge, but this is scarcely valid when other practitioners are showing that they can cure the common neuroses in about the same time as is required for the average surgical or obstetrical case. There are some patients who do require months or years of patient treatment and who are beyond our capabilities; but these are a minority.

The conditions in general practice are not always ideal for psychiatric work; but the other advantages and our duty to our patients outweigh this.

Comments.

Thorner in America, Watts in England, and Chapman, pioneering the field in Australia, have recently demonstrated in reports of their own clinical work that most neuroses can be, and should be, treated in general practice. It has been the purpose of this article to present further confirmation and to show in some detail how this can be done. It is hoped that the record of my experiences may encourage others and enable them to avoid some of the pitfalls.

A few fortunates are born psychiatrists and do and say the right things intuitively; but most of us have to cultivate this art. This requires some basic knowledge, but mainly personal application and practice in learning to listen in a different manner from "history-taking", in observing carefully the bodily effects of repressed emotion, in practising the art of releasing these, and, above all, in learning to see the patient not only as he is, but also as he may become after such release.

There is one pitfall in psychiatric work which can probably be avoided only by the fortunate few. This is described in Balint's article. One must "face the experience that quite often one's actual behaviour is entirely different from what one has always believed it to be". "It is a fact that acquiring psychotherapeutic skill is tantamount to discovering some, hard, and not very pleasant, facts about one's own limitations."

Most, if not all, practitioners are aware of the prevalence of, and the problems created by, neuroses. Most practitioners sincerely wish to face their responsibilities and to cure these patients. But few apparently make any serious effort to treat them (other than by sedatives or tonics, reassurance and advice), even though others show them that this can be done. This is curious, but perhaps explicable by Balint's article, from which the foregoing extracts are taken.

The process of becoming aware of one's automatic patterns and acquiring a modicum of freedom from them has been, and still is, painful at times; but there is a deeper satisfaction in learning to know oneself (or one's selves) and in being able effectively to treat the substantial proportion of patients whose sufferings derive from psychic causes.

Group therapy, such as Balint describes, does appear to be an excellent method of learning to acquire skill in psychiatric diagnosis and treatment; but, until such time as it comes into more general application, such awareness and skill can be acquired by patient and persevering personal effort. Whenever it can be arranged, as I have been able to do recently, frank discussion with one or more colleagues will help considerably.

Summary.

A practical method of approach to the diagnosis and treatment of neuroses in general practice is presented. Case histories are quoted to illustrate the common difficulties and errors of diagnosis, and a method of preventing or minimizing these. One method of treatment is described and the results of such treatment are given.

Further evidence is submitted to show that the general practitioner should treat most of the neuroses that he encounters commonly, and that this can be done successfully.

Acknowledgements.

I wish to express my gratitude to the authors whom I have mentioned, and to my patients, who have encouraged and taught me, often knowing that I was a novice in a new and imperfectly explored territory.

References.

- BALINT, M. (1954), "Training General Practitioners in Psychotherapy", *Br. M. J.*, 1: 115.
 CHAPMAN, H. O. (1953), "Neurosis in General Practice", *M. J. AUSTRALIA*, 2: 407.
 HADFIELD, J. A. (1952), "Psychology and Mental Health", Allen and Unwin.
 KENNEDY, R. S. (1953), "The Beattie-Smith Lectures", *M. J. AUSTRALIA*, 2: 625 and 661.
 ROGERS, C. L. (1942), "Counselling and Psychotherapy", Houghton Mifflin, New York.
 ROSS, T. A. (1942), "The Common Neuroses", Arnold, London.
 THORNER, M. W. (1948), "Psychiatry in General Practice", Saunders.
 WATTS, C. A. H., and WATTS, B. M. (1952), "Psychiatry in General Practice", Churchill, London.

AN ELECTROLYTE CONVERSION TABLE.

By J. A. C. DIQUE,

Department of Pathology, Brisbane General Hospital, Brisbane.

WITH the growth of intravenous fluid therapy today, it has become increasingly important to estimate the concentration of tissue electrolytes in milliequivalents per litre instead of in milligrammes per 100 millilitres. All workers in this field agree that this facilitates assessment of the tissue concentrations of electrolytes and leads to more accurate prescription with respect to quantities to be replaced.

The absence of a ready reckoner for converting milligrammes per 100 millilitres of crystalloid, grammes per 100 millilitres of protein or volumes *per centum* of carbon dioxide into milliequivalents per litre has undoubtedly played its part in hindering the use of the milliequivalents per litre system on account of the mere labour of making the required calculations.

It is with the intention of filling this gap that I introduce the table below. By means of this table it is possible to convert milligrammes of crystalloid per 100 millilitres, grammes per 100 millilitres of protein, and volumes *per centum* of carbon dioxide into milliequivalents per litre by merely adding three numbers together.

I once constructed a series of nomograms and from them a complete list of milliequivalents per litre values for all known and likely concentrations of electrolytes. There were eight pages of nomograms and ten pages of type-written figures, a volume of paper far too bulky when the information can be obtained from one sheet of paper.

Construction of the Table.

A brief note of how the table was constructed would be informative.

The concentration of a substance in milliequivalents per litre may be calculated in the following way:

1. Milliequivalents of crystalloid per litre =

$$\frac{\text{Milligrammes of substance per 100 millilitres} \times 10 \times \text{valency}}{\text{Molecular weight.}}$$
2. Milliequivalents of base bicarbonate per litre =

$$\frac{\text{Carbon dioxide combining power}}{2.24}$$

3. Base binding power of serum protein =

$$\frac{\text{Grammes per 100 millilitres of protein} \times 2.43}{\text{Base binding power of serum protein}}$$

By the use of these formulae and taking the atomic weights and valencies of tissue electrolytes as under, I have calculated the concentration of tissue electrolytes in

TABLE I.

Grammes of Protein per 100 millilitres; Volumes per Centum of CO ₂ ; Milligrammes per 100 millilitres Crystalloid.	Electrolytic Concentration in Milliequivalents per Litre to the Nearest Tenth of a Milliequivalent.									
	Cl-If Expressed as NaCl.	Cl-If Expressed as Chlorine.	HCO ₃ .	Protein.	HPO ₄ .	-SO ₄ .	Na.	K.	Ca.	Mg.
100	17.1	28.2	44.6	243.0	18.8	20.8	43.5	25.5	49.9	82.2
200	34.2	56.4	89.3	486.0	37.5	41.6	87.0	51.0	99.8	164.5
300	51.3	84.6	133.9	729.0	56.3	62.5	130.5	75.5	149.7	246.7
400	68.4	112.8	178.6	972.0	75.0	83.8	173.9	102.0	199.6	328.9
500	85.5	141.0	223.2	1215.0	93.8	104.1	217.4	127.5	249.5	411.2
600	102.6	169.2	267.9	1458.0	112.5	124.9	260.9	153.0	299.4	493.4
700	119.7	197.4	312.5	1701.0	131.3	145.7	304.4	178.5	349.3	575.7
800	136.8	225.6	357.1	1944.0	150.0	166.6	347.9	204.0	399.2	657.9
900	154.0	253.8	401.8	2187.0	168.8	187.4	391.4	230.0	449.1	740.1

milliequivalents per litre for values of 100 to 900 milligrammes per 100 millilitres in the case of crystalloid, volumes *per centum* for carbon dioxide, and grammes per 100 millilitres for protein. Calculations have been made only for exact hundreds. The concentration in milliequivalents per litre of numbers that are not exact hundreds is obtained by moving the decimal point and adding up the units, tens and hundreds which go to make up that number.

It must be pointed out that whereas a carbon dioxide combining power of 600 volumes *per centum* having a base bicarbonate concentration of 267.9 milliequivalents per litre cannot occur in Nature, it is nevertheless an accurate means of expressing the fact that a figure of 60 volumes of carbon dioxide *per centum* has a base bicarbonate concentration of 26.79 milliequivalents per litre. Similarly a figure such as 700 grammes of protein per 100 millilitres (an impossible figure) with a concentration equivalent to 1701.0 milliequivalents per litre is really a means of expressing the fact that seven grammes *per centum* of protein have a concentration of 17.01 milliequivalents per litre.

The numbers 100 to 900 in the first column of the table refer to the concentration of the various substances—grammes per 100 millilitres for protein, volumes *per centum* for carbon dioxide combining power, and milligrammes per 100 millilitres for the other substances. Each number in each of the other columns represents the concentration in milliequivalents per litre of the substance at the head of the column to the nearest tenth of a milliequivalent.

The method of using the chart can most easily be understood from the following examples:

1. The serum sodium content is 281 milligrammes per 100 millilitres; what is its concentration in milliequivalents per litre?

By reference to the chart:

200 milligrammes of sodium per 100 millilitres have a concentration of 87.0 milliequivalents per litre.

80 milligrammes of sodium per 100 millilitres have a concentration of 34.79 milliequivalents per litre.

1 milligramme of sodium per 100 millilitres has a concentration of 0.435 milliequivalent per litre.¹

So by addition:

281 milligrammes of sodium per 100 millilitres have a concentration of 122.2 milliequivalents per litre.

2. The serum protein content is 6.8 grammes per 100 millilitres; what is its base binding power in milliequivalents per litre?

¹ Obtained by moving the decimal point.

By reference to the chart:

6.0 grammes of protein per 100 millilitres have a concentration of 14.58 milliequivalents per litre.

0.8 gramme of protein per 100 millilitres has a concentration of 1.944 milliequivalents per litre.

So by addition:

6.8 grammes of protein per 100 millilitres have a concentration of 16.5 milliequivalents per litre.

TABLE II.

Substance.	Atomic Weight.	Valency of Ion.
Na	22.997	1
K	39.098	1
Mg	24.32	2
Ca	40.08	2
S	32.06	2 (of SO ₄)
P	30.98	1.8 (of HPO ₄) ¹
Cl	35.457	1
O	16.000	2
C	12.010	1 (of HCO ₃)
H	1.008	1

¹ The valency varies with the pH. At a pH of 7.4, HPO₄ unites with 1.8 equivalents of base.

3. What is the base bicarbonate concentration in milliequivalents per litre of a specimen of serum having a carbon dioxide combining power of 43.5 volumes *per centum*?

By reference to the chart:

40.0 volumes of carbon dioxide *per centum* have a concentration of 17.86 milliequivalents per litre.

3.0 volumes of carbon dioxide *per centum* have a concentration of 1.339 milliequivalents per litre.

0.5 volume of carbon dioxide *per centum* has a concentration of 0.2232 milliequivalent per litre.

So by addition:

43.5 volumes of carbon dioxide *per centum* have a concentration of 19.4 milliequivalents per litre.

The concentration of chlorine is sometimes expressed as milligrammes of chlorine per 100 millilitres; more usually it is expressed as milligrammes of sodium chloride per 100 millilitres. If the concentration is expressed in milligrammes of chlorine per 100 millilitres, then use the first and third columns. If, however, the concentration is expressed as milligrammes of sodium chloride per 100 millilitres, the first and second columns should be used in order to work out the concentration of chlorine in milliequivalents per litre. The first and second columns may also be used to determine the number of millimoles of sodium and chlorine per litre contained in routine sodium

chloride solutions—for example, 0.9 gramme of sodium chloride *per centum* has a concentration of 154 milliequivalents of sodium and chloride each per litre.

The concentration of substances in milliequivalents per litre is accurate to the nearest tenth of a milliequivalent for all substances in which the concentrations in grammes, milligrammes and volumes of carbon dioxide are exact hundreds.

Should it be desired to obtain the concentration of a substance in millimoles per litre, this can be readily done by dividing the concentration in milliequivalents per litre by the valency of the substance.

For proteins divide the concentration in milliequivalents per litre by eight.

Patients who are being maintained on intravenous therapy and who are known or thought to be deficient in potassium should have this ion replaced intravenously. As the salt usually chosen is potassium chloride, it is useful to know how many milliequivalents are contained in a fixed quantity of salt. The same is true of calcium. The following figures are worth remembering:

1. Potassium chloride, 1.0 gramme
= 475.6 milligrammes of chlorine + 524.4 milligrammes of potassium
= 13.4 milliequivalents of chlorine and potassium each.
2. Calcium chloride, 1.0 gramme
= 361.1 milligrammes of calcium + 638.9 milligrammes of chlorine
= 18.0 milliequivalents each.

Summary.

An electrolyte conversion table has been presented and the manner of using it described.

References.

- BLAND, J. H. (1952), "The Clinical Use of Fluid and Electrolyte", Saunders, Philadelphia.
- GAMBLE, J. L. (1952), "Chemical Anatomy, Physiology and Pathology of Extracellular Fluid", 5th Edition, Harvard University, Cambridge, Massachusetts.
- MARRIOTT, H. L. (1950), "Water and Salt Depletion", Blackwell, Oxford.
- Medical Research Council Memorandum Number 26 (1953), "The Treatment of Acute Dehydration in Infants", by a working team appointed and advised by the Committee on Acute Infections in Infancy, Her Majesty's Stationery Office, London.
- MOYER, C. A. (1952), "Fluid Balance", Yearbook Publishers, Chicago.
- NEUBURGH, L. H. (1951), "Significance of Body Fluids in Clinical Medicine", Thomas, Springfield.

POST-GASTRECTOMY DIETARY MANAGEMENT.¹

By PATRICIA J. CROWE,

Research Dietitian, Clinical Research Unit,
Royal Prince Alfred Hospital,

AND

STANLEY GOULSTON,
Sydney.

The operation of partial gastrectomy is at present the standard procedure when surgery is indicated for gastric and duodenal ulceration. The operation usually cures the patient, but he may suffer in other ways as a direct result of the altered physiology occasioned by the operation.

As a rule, physicians play only a small part in the post-operative management of these patients, and surgeons are concerned mainly with their patients' recovery from the operation. We have found that much confusion exists as to dietary measures recommended after gastrectomy.

¹ From the Gastro-enterological Clinic, Royal Prince Alfred Hospital, Sydney.

Surgeons differ in their outlook in this regard. Some tell their patients to eat and drink anything, others instruct them to remain on an ulcer type of diet indefinitely. Some patients receive no instructions, and these often resort to a diet consisting mainly of carbohydrate. Women are the main offenders.

The post-gastrectomy syndromes commencing soon after the operation have in recent years been more clearly defined into three main entities: the "efferent loop syndrome", in which the essential feature is the rapid entry of a large bulk of food into the jejunum; the "afferent loop stasis syndrome", due to distension of the afferent loop caused by mechanical kinking at its junction with the gastric remnant; and the "gastric distension syndrome", due to slow emptying of the stomach with a narrow or defective stoma. These syndromes cause symptoms during or at the end of the meal, such as fullness in the upper part of the abdomen, consciousness of intestinal movement, vomiting of bile or food, diarrhoea, and vasomotor phenomena such as sweating, palpitation, pallor, feeling of warmth, tachycardia and excessive tiredness. These inconvenient symptoms may be transient, but may persist for long periods after operation. Their incidence has been described as between 5% and 12% (Custer, Butt and Waugh, 1946).

TABLE I.

Failure to Gain Weight after Gastrectomy.

Authority.	Year.	Number of Cases.	Condition.
Sara Jordan ..	1941.	100	29% below normal weight after one year.
Adlersberg and Hammerschlag	1946 } 1949 }	—	Average loss, 10 pounds after seven years.
Muir ..	1949	86	40% lost seven pounds or more, less than 10% regained weight, six months after operation.
Wells and Welbourn.	1951	100	Average loss, 14 pounds one year after gastrectomy.

Adlersberg and Hammerschlag in 1947 separated these post-gastrectomy symptoms from those occurring months later and due to hypoglycaemia.

In addition to these syndromes a number of patients suffer from troublesome gastritis in the gastric remnant, beginning in the first weeks after the operation and recurring thereafter. A small percentage of the duodenal ulcer sufferers who undergo operation will develop a stomal ulcer.

Furthermore, post-gastrectomy patients may fall to gain weight and may remain debilitated owing to a diminished caloric intake and impaired digestion and absorption of food. They may be unable to resume their former occupation or to attempt heavy work owing to lack of strength and vitality. Evidence has been presented that pulmonary tuberculosis occurs more commonly after partial gastrectomy than in patients with an intact stomach.

In our opinion the post-gastrectomy syndromes, malnutrition and debility, could be prevented or minimized by close attention to the patient's diet during the first three months after operation. The gastric remnant with an artificial stoma requires education in its handling of food-stuffs, and intelligent dietary methods should be undertaken from the first week after gastrectomy. The diet should contain sufficient calories to result in a weight gain from the outset.

The diet regimen should be based on the following considerations: (a) The early introduction of sufficient calories and protein to combat the post-operative negative nitrogen and potassium balance. (b) A gradual but definite increase in food intake each week. (c) Division of the intake into six small feeds daily until the end of the third month, by which time the patient's pre-ulcer diet can usually be taken. The six small meals offer practical difficulties in hospital because of the extra work entailed for the nursing and kitchen staffs, but are essential in the patient's gastric reeducation. The meals should be

attractively served. (d) The giving of clear directions concerning the type of foods the patient is advised to eat on leaving hospital to introduce variety and to satisfy personal and racial preferences.

TABLE II.
Types of Food Patient is Advised to Eat till End of Third Month.

Food.	Advised.	To be Avoided.
Meat.	Tender red or white meats, such as grilled lamb chops or cutlets, roast lamb, roast beef, roast veal, roast and creamed chicken, roast and creamed rabbit, creamed brains, Crisp bacon.	Tough cuts, made-up meat dishes such as rissoles, sausages, frankfurts, highly seasoned meat, stews, gravies, curries, seasoned, smoked, canned meats.
Fish.	Creamed and steamed fish, varieties such as flathead, bream, butterfish, schnapper, whiting <i>et cetera</i> , salmon, sardines, oysters, whitebait.	Crab, lobster, prawns.
Eggs.	Poached, boiled, scrambled omelettes.	Fried.
Cheese.	All types of cheese—for example, mild, cheddar, tasty, Roquefort, Camembert. Cheese dishes—for example, cheese omelette, cheese fondue, <i>et cetera</i> .	
Entrées.	Macaroni, spaghetti, rice.	Baked beans, sweet corn.
Vegetables.	Tender young vegetables such as beans, peas, carrots, pumpkin, marrow, chokoes, squash, spinach, asparagus, cauliflower, potatoes.	Cabbage, onions, parsnips, Brussels sprouts, turnips.
Salads.	Tender young inside leaves of lettuce, tomatoes, beetroot.	Cucumber, celery, radish.
Fruits and Fruit Juices.	All stewed and canned fruits, ripe bananas, peaches and pears. Dried fruits. Fruit juices—for example, orange, lemon, pineapple and tomato.	Unripe fruits, pineapple.
Bread and Cereals.	Porridge, cornflakes, rice bubbles, "Weetbix". Day old white or brown bread.	Fresh bread, whole wheat bread, cereals with bran.
Desserts.	Preferably made with eggs and milk—for example, baked rice custard, Spanish cream, blanc-mange, lemon delicious, gelatine desserts, rice, sago, tapioca, junket, light steamed sponge, sponge cake or plain butter cake, fruits listed above, light steamed puddings.	Pies, pastries, hot scones, heavy steamed fruit puddings, spices, rich fruit cake.
Beverages.	Milk coffee, weak tea with milk or lemon, soft drinks. Milk, 1 to 1½ pints per day. "Aktavite", cocoa <i>et cetera</i> .	Avoid all alcohol for first three months.
Nuts.	—	Not allowed till end of three months.
Condiments.	Salt, tomato purée, lemon juice, "Marmite", "Vegemite", salad dressing, French salad dressings in small amounts.	Curry, mustard, pickles, pepper, tomato sauce, vinegar, Worcestershire sauce.
Jams, Sweets <i>et cetera</i> .	Sugar, honey, syrups, jellied jams, jams without skins or pips. Plain chocolate, boiled lollies, butterscotch <i>et cetera</i> .	Chocolate with nuts.
Fats.	Butter, cream, oil. Introduce foods fried in minimum of fat gradually towards end of three months period.	—

The following dietary programme has been compiled to conform with these principles.

Suggested Feeding Schedules.¹

First Week.

The patient who has had a gastrectomy will have an indwelling tube in the stomach proximal to the anastomosis through which gastric contents are aspirated for one to

¹ All protein, fat and carbohydrate contents are in grammes.

three days. Provided there are no complications such as gastric dilatation or electrolyte imbalance, the tube is then removed. Feeding during the first two to three days is by the intravenous route under the surgeon's care. After twelve to twenty-four hours small sips of water are allowed by mouth. A useful schedule is as follows:

Thirty-six to forty-eight hours after operation: One ounce of water hourly during waking period.

Day 3: One ounce of citrated milk two-hourly during waking period.

Day 4: Two ounces of citrated milk two-hourly during waking period.

Day 5: Three ounces of citrated milk two-hourly during waking period.

Day 6: Six ounces of citrated milk two-hourly during waking period.

Day 7: Six ounces of citrated milk two-hourly during waking period, with mashed potato, junket, custard, jelly *et cetera*.

If milk is badly tolerated or disliked, the following fluid alternative is suggested: strained fruit juices (for example, orange, lemon), to which unbeaten egg white has been added in the proportion of one egg white to four ounces of fruit juice.

Second Week.

Protein, 77 grammes; fat, 93 grammes; carbohydrate, 141 grammes. Calories, 1709.

Protein. Fat. Carbo-
hydrate.

7 a.m.:

6 ounces milk, or milk coffee,
cocoa, "Milo" 6 7 7

Calories, 115.

8 a.m.:

1 egg 6 6 —
½ slice bread 1 — 3
1 teaspoon butter — 3 —
1 teaspoon honey — — 4
6 ounces milk, or milk coffee,
cocoa, "Milo" 6 7 7

13 16 19

Calories, 272.

10 a.m.:

½ slice bread 1 — 8
1 teaspoon butter — 3 —
Honey or jam — — 4
6 ounces milk, or milk coffee,
cocoa, "Milo" 6 7 7

7 10 19

Calories, 194.

12 midday:

Steamed fish (1 serve), or chicken,
or rabbit 15 3 —
2 tablespoons white sauce or
gravy 2 4 7
3 tablespoons potato 2 — 18
6 ounces orange juice 1 — 15

20 7 40

Calories, 303.

3 p.m.:

Ice cream, 1 small bucket 1 3 5
Stewed fruit (1 serve) 1 — 11
1 ounce cream 1 10 1
6 ounces milk, or milk coffee,
cocoa, "Milo" 6 7 7

9 20 24

Calories, 312.

5 p.m.:

Omelette—1 egg 7 9 2
½ slice bread 1 — 8
1 teaspoon butter — 3 —
6 ounces milk, or milk coffee,
cocoa, "Milo" 6 7 7

14 19 17

Calories, 295.

7 p.m.:

6 ounces milk cocoa 7 9 8
1 "Sao" biscuit 1 2 7
1 teaspoon butter — 3 —

8 14 15

Calories, 218.

Third Week.

Protein, 100 grammes; fat, 90 grammes; carbohydrate, 164 grammes. Calories, 1866.

Skim milk mixture: 1½ pints milk, 4 ounces dried skim milk, flavoured.

	Protein.	Fat.	Carbo- hydrate.
7 a.m.:			
6 ounces milk	6	7	7

Calories, 115.

8 a.m.:			
1 egg	6	6	—
1 slice bread	1	—	8
2 teaspoons butter	—	5	—
1 teaspoon honey, or "Marmite"	—	—	3
6 ounces milk mixture	10	5	13
	17	16	24

Calories, 308.

10 a.m.:			
1 slice bread, or 1 or 2 "Sao" biscuits	1	—	8
2 teaspoons butter	—	5	—
1 inch cube cheese	4	—	—
6 ounces milk mixture	10	5	13
Honey or jam	—	—	3
	15	15	24

Calories, 291.

1 p.m.:			
2 slices (1½ ounces) tender red or white meat	9	10	—
2 tablespoons white sauce or gravy	2	4	7
2 tablespoons potato	1	—	12
2 tablespoons pumpkin	1	—	6
6 ounces orange juice	1	—	15
	14	14	40

Calories, 342.

3 p.m.:			
Baked custard (1 serve)	6	6	10
Stewed fruit (1 serve) and sugar	1	—	11
6 ounces milk mixture	10	5	13
	17	11	34

Calories, 303.

5 p.m.:			
Creamed soup or chicken broth	4	5	1
1 slice (1 ounce) meat	6	7	—
1 slice bread	1	—	8
2 teaspoons butter	—	5	—
6 ounces milk mixture	10	5	13
	21	22	22

Calories, 370.

7 p.m.:			
6 ounces milk mixture	10	5	13

Calories, 137.

Fourth and Fifth Weeks.

Protein, 96 grammes; fat, 138 grammes; carbohydrate, 196 grammes. Calories, 2410.

7 a.m.:			
1 cup cereal	2	1	17
2 teaspoons sugar	—	—	10
6 ounces milk	2	2	2
Stewed fruit (1 serve) and sugar	1	—	11
1 cup milk (6 ounces)	6	7	7
	11	10	47

Calories, 322.

9 a.m.:			
1 egg	6	6	—
1 slice bread	2	—	16
3 level teaspoons butter	—	5	—
6 ounces milk, as coffee, cocoa	6	7	7
	14	18	23

Calories, 310.

11 a.m.:

1 egg flip—			
1 egg	6	6	—
6 ounces milk	6	7	7
1 ounce cream (2 table- spoons)	1	10	1
Vanilla	—	—	—
1 "Sao" biscuit	1	2	7
1 teaspoon butter	—	3	—
1 ounce cheese	4	5	—
	18	33	15

Calories, 429.

1 p.m.:			
2 slices (1½ ounces) white or red meat	9	12	—
2 tablespoons white sauce or gravy	2	4	7
3 tablespoons mashed potato	2	—	18
Beans (1 serve) or equivalent	1	—	6
1 glass (6 ounces) orange juice	1	—	15
	15	16	46

Calories, 388.

3 p.m.:			
Protein dessert—for example, Spanish cream, baked custard, lemon delicious	6	6	15
2 tablespoons (1 ounce) cream	1	10	1
1 slice bread	1	—	8
1 teaspoon butter	—	3	—
6 ounces milk, or cocoa, coffee	6	7	7
	14	26	31

Calories, 414.

6 p.m.:			
Cheese omelette	10	16	—
1 slice bread	2	—	16
2 teaspoons butter	—	5	—
1 teaspoon honey, "Marmite" or jam	—	—	4
6 ounces milk, as "Bournvita", coffee	6	7	7
Tea	—	—	—
	18	28	27

Calories, 432.

9 p.m.:			
6 ounces milk, as "Ovaltine", cocoa, coffee	6	7	7

Calories, 115.

Sixth to Twelfth Weeks.

Protein, 102 grammes; fat, 123 grammes; carbohydrate, 254 grammes. Calories, 2531.

Breakfast:			
1 cup cereal	2	1	17
2 teaspoons sugar	—	—	10
2 ounces milk	2	2	2
1 egg	6	6	—
Bacon, 1 rasher (5 inches)	4	4	—
1 slice bread	2	—	16
2 teaspoons butter	—	5	—
2 teaspoons jam	—	—	14
Coffee or tea	—	—	—
	16	18	59

Calories, 462.

Morning tea:			
6 ounces milk	6	7	7
2 "Sao" biscuits	2	3	13
2 teaspoons butter	—	5	—
1 ounce cheese	4	5	—
	12	20	20

Calories, 308.

2 slices bread	4	—	31
3 teaspoons butter	—	8	—
2 slices cold roast meat or 2 ounces white meat—for example, fish	12	14	—
Lettuce, tomato or hot vege- tables—for example, potato, pumpkin, beans	—	—	3
1 piece (6 ounces) fruit—for example, ripe pear, orange	1	—	15
6 ounces milk	6	7	7
	23	29	56

Calories, 577.

	Protein.	Fat.	Carbo- hydrate.
Afternoon tea:			
6 ounces milk	6	7	7
1 piece (2 ounces) cake	5	4	30
	11	11	37

Calories, 291.

Dinner:			
4 ounces grilled steak	24	28	—
3 tablespoons potato	2	—	18
Peas (1 serve)	3	—	17
Pumpkin (1 serve)	1	3	6
Ice cream (one scoop)	1	—	13
Stewed fruit and sugar, or fresh fruit	1	—	13
Tea.	31	31	54

Calories, 619.

Supper:			
6 ounces milk cocoa	7	9	8
1 slice toast	2	—	16
2 teaspoons butter	—	5	—
Honey or jam or "Marmite"	—	—	4
	9	14	28

Calories, 274.

References.

- ADLERSBERG, D. and HAMMERSCHLAG, E. (1947), "The Post Gastrectomy Syndrome", *Surgery*, 21: 720.
- CUSTER, M. D., BUTT, R., and WAUGH, J. M. (1946), "So-called Dumping Syndrome after Gastrectomy", *Ann. Surg.*, 123: 410.
- JORDAN, S. (1941), "End Results of Radical Surgery of the Gastrointestinal Tract", *J.A.M.A.*, 116: 586.
- MUIR, A. (1949), "Post Gastrectomy Syndrome", *Brit. J. Surg.*, 37: 165.
- WELLS, C., and WELBOURN, R. (1951), "Post Gastrectomy Syndrome", *Brit. M. J.*, 1: 546.

THE PATTERN OF CONGENITAL HEART DISEASE IN INFANCY AND CHILDHOOD.

By DOUGLAS STUCKEY,

Physician-in-Charge, Congenital Heart Disease Clinic,
Royal Alexandra Hospital for Children, Sydney.

THIS report is based on experience at the Royal Alexandra Hospital for Children, Sydney, from May, 1948, to December, 1953. Three hundred and thirty-six patients suffering from a congenital heart lesion were examined at the Congenital Heart Disease Clinic during this period. In 19 of these cases the results of post-mortem examination were available, in 57 the diagnosis was confirmed at operation, and in 69 either angiocardiology or cardiac catheterization had been carried out. In the remainder, the diagnosis was based on repeated clinical examination and assessment by the members of the Congenital Heart Disease Clinic, together with radiological examination including fluoroscopy, a standard twelve-lead electrocardiogram and a full blood count. In the same period, the diagnosis of a normal heart with an innocent systolic murmur was made in 58 cases at the clinic, and this group of patients will be reported elsewhere.

During the same period of time, post-mortem examination revealed a congenital heart lesion in an additional 90 patients who had died in the wards of the hospital and who had not been examined at the Congenital Heart Disease Clinic. This happened when death occurred within a few days of admission to hospital, or when the child was admitted to hospital with some other illness and the presence of a congenital heart lesion was not suspected during life. Of the 109 children on whom a post-mortem examination was carried out, 71 were aged under six months (nine of these died in the first two weeks of life), 26 were aged between six months and two years, and 12 were aged between two and thirteen years.

The incidence of the various forms of congenital heart disease amongst all the patients with a congenital malformation of the heart was recorded in two age groups—the first from birth up to the second birthday (204 patients), and the second from the age of two years to the thirteenth birthday (222 patients). The incidence of the different forms of congenital heart disease in these two age periods is presented in Table I.

TABLE I.

Type of Heart Disease.	Up to Two Years. (204 Patients.)	Two to Thirteen Years. (222 Patients.)
<i>Acyanotic forms of congenital heart disease:</i>		
Patent ductus arteriosus	5.0%	30.0%
Atrial septal defect	10.0%	12.0%
Ventricular septal defect	20.0%	20.0%
Pulmonary stenosis	5.0%	10.0%
Aortic stenosis	0.5%	3.0%
Coarctation of the aorta (adult type) ..	1.0%	1.5%
Atrio-ventricularis communis	5.0%	—
Other acyanotic forms	5.0%	1.0%
<i>Cyanotic forms of congenital heart disease:</i>		
Tetralogy of Fallot	15.0%	15.0%
Pulmonary atresia	5.0%	1.0%
Pulmonary stenosis with reversed inter- atrial shunt	1.5%	0.5%
Tricuspid atresia	5.0%	0.5%
Eisenmenger group	5.0%	4.0%
Transposition of the great vessels ..	10.0%	0.5%
Truncus arteriosus	2.0%	1.0%
Coarctation of the aorta (infantile type)	5.0%	—
Total	100.0%	100.0%

¹ Angiocardiography or cardiac catheterization was carried out in nine cases in the first group and in 60 cases in the second group; no operations were performed in the first group, but 57 subjects in the second group were operated on; post-mortem examinations were carried out on 97 subjects in the first group and on 12 subjects in the second group.

The classification used was essentially a clinical one. In the Eisenmenger group, as suggested by Wood (1952), were included not only patients with a true Eisenmenger's complex, an uncommon anomaly which can be recognized with certainty only at post-mortem examination, but also all patients with severe pulmonary hypertension and a reversed flow of blood from the right to the left side of the heart through a shunt—a patent ductus arteriosus, an atrial septal defect or a ventricular septal defect. The last-mentioned conditions present an almost identical clinical picture, and the distinction between them can be made only with difficulty during life. The disordered physiology is very similar and they have the same treatment, course and prognosis, so that it is helpful to consider these patients as one group from the clinical point of view. Pulmonary stenosis with reversed interatrial shunt is a clinical diagnosis depending on the presence of persistent central cyanosis during life in a patient with severe pulmonary stenosis, an intact interventricular septum and a patent foramen ovale. In the remainder the clinical and pathological diagnoses corresponded.

When multiple defects were present, the patient was classified under the one of major importance. For instance, all patients with tricuspid atresia or pulmonary stenosis with reversed interatrial shunt, and some with a *truncus arteriosus*, transposition of the great vessels or the tetralogy of Fallot, have a defect in the interatrial septum; but this is secondary to, or incidental to, the major anomaly. Slit patency of the foramen ovale, which as an isolated post-mortem finding has no functional significance, was ignored, as was a patent ductus arteriosus in a patient aged under six weeks, unless it was very large.

Discussion.

Some knowledge of the relative frequency of the various congenital malformations of the heart at different ages is extremely helpful in clinical diagnosis.

Maude Abbott's (1915) anatomical classification of 1000 cases, while useful for reference, was based on post-mortem

material only, and is too complex to have much clinical application. Wood (1950) reported incidence figures from a clinical point of view; but his series included many adults and very few children aged under three years.

In childhood, from the ages of two to thirteen years, the incidence figures presented in this paper agreed well with those reported by Wood (1950). Cyanotic forms of congenital heart disease represented about one-fifth of all cases, and two out of three of these patients had the tetralogy of Fallot, so that this malformation dominated all others in importance in the child aged over two years with cyanotic congenital heart disease. Of the acyanotic forms, patent *ductus arteriosus*, atrial septal defect, ventricular septal defect and pulmonary stenosis between them accounted for about nine out of ten cases. It would be expected that in older patients the organic stenoses (aortic stenosis, pulmonary stenosis, coarctation of the aorta), which remain relatively fixed in size during the growth of the child, would become more important in relation to increasing body size, and that some patients with these conditions would present for the first time in adolescence or early adult life.

In infancy, up to the age of two years, the incidence pattern of the various forms of congenital heart disease was strikingly different. Cyanotic forms of congenital heart disease accounted for almost half of all patients examined, owing to the greater numbers of patients suffering from transposition of the great vessels, pulmonary atresia, tricuspid atresia, and the infantile form of coarctation of the aorta. The tetralogy of Fallot was still the commonest form of cyanotic congenital heart disease found, but accounted for less than one-third of the cases of cyanotic congenital heart disease in this age group. During the period under review, 13 patients aged under two years suffering from the tetralogy of Fallot died.

With regard to the patients with acyanotic forms of congenital heart disease in infancy, there were several points of interest. Patent *ductus arteriosus* was recognized infrequently in this age group; of 76 patients with this malformation examined at the clinic, the diagnosis was made on clinical grounds only twice in subjects aged under two years. During the same period, seven infants aged under two years with a patent *ductus arteriosus*, who had not been examined at the clinic, died in the wards of the hospital, and the anomaly was recognized at post-mortem examination. It would seem that this essentially curable lesion is sometimes overlooked in infancy, and the possibility should be considered in relation to any infant with congestive heart failure of uncertain origin. Persistent *atrio-ventricularis communis* was encountered only in this age group; of 14 patients with this anomaly at post-mortem examination, the eldest was aged twelve months. The organic stenoses (aortic stenosis, pulmonary stenosis, coarctation of the aorta) were recognized less frequently in infancy. A possible explanation for this has already been mentioned.

The sex incidence in particular forms of congenital heart disease is of some importance. Thus with patent *ductus arteriosus*, females predominated in the proportion of more than three to one (56:17), and conversely there was strong male preponderance in coarctation of the aorta, especially the adult type (6:0), but also in the infantile form (7:2). These tendencies in sex incidence have been noted before (Brown, 1950). Wood's (1950) figures agreed in the case of patent *ductus arteriosus*, but not in coarctation of the aorta, in which he found equal sex incidence. In atrial septal defect, female preponderance was found to be slight (27:20). In the cyanotic forms of congenital heart disease there were no outstanding differences in sex incidence.

It is apparent that there is a considerable loss of life from congenital heart disease in infancy, and that a proportion of the children who die have a malformation potentially amenable to surgery. It is probable that, in the future, an increasing number of surgical operations on these children will be undertaken in the first few years of life. For this reason, infants and children should be

referred for an opinion or further investigation whenever the possibility of congenital heart disease is suspected.

Summary.

The incidence of the various forms of congenital heart disease among 426 patients with a congenital malformation of the heart examined at the Royal Alexandra Hospital for Children, Sydney, since 1948, is presented in two age groups.

In the childhood age group, from two to thirteen years of age, cyanotic forms of congenital heart disease accounted for one-fifth of the cases, and in two-thirds of these the condition was the tetralogy of Fallot. Of the acyanotic forms, patent *ductus arteriosus*, atrial septal defect, ventricular septal defect and pulmonary stenosis between them accounted for nine out of ten cases.

In the age period of infancy, from birth up to two years of age, the incidence pattern was strikingly different. Cyanotic forms of congenital heart disease accounted for almost half the cases, because of the greater numbers of patients suffering from transposition of the great vessels, pulmonary atresia, tricuspid atresia and the infantile type of coarctation of the aorta. Of the acyanotic forms, patent *ductus arteriosus* was recognized infrequently in this age period, and one patient in 10 had a persistent *atrio-ventricularis communis*.

Attention is drawn to the increasing importance of the diagnosis and treatment of congenital heart lesions at an early age.

Acknowledgements.

The other members of the Congenital Heart Disease Clinic, medical, surgical and radiological, have contributed a great deal to this study. In particular a tribute should be paid to the late Dr. Stanley G. Bradfield, who died tragically in 1951, and whose energy and enthusiasm were largely responsible for the successful functioning of the clinic from its early days.

References.

- ABBOTT, M. E. (1915), "Congenital Cardiac Disease", in Osler and McCrae's "Modern Medicine", 2nd Edition, Lea and Febiger, Philadelphia, 4: 323.
- BROWN, J. W. (1950), "Congenital Heart Disease", 2nd Edition, Staples Press, London: 30.
- WOOD, P. (1950), "Congenital Heart Disease", *Brit. M. J.*, 2: 639.
- WOOD, P. (1952), "Pulmonary Hypertension", *Brit. M. Bull.*, 8: 348.

Reviews.

Diseases of the Knee: Management in Medicine and Surgery.
By Anthony F. DePalma, M.D.: 1954. Philadelphia: J. B. Lippincott Company. Sydney: Angus and Robertson, Limited. 10½" x 7½", pp. 862, with 455 illustrations. Price: £10 15s.

TEXT-BOOKS of orthopaedic surgery are usually written for the undergraduate or are so superficial that they can lead the occasional orthopaedic surgeon into trouble. This second book by Professor DePalma follows his volume on the shoulder and is complete enough for the post-graduate student and for the practising surgeon, either general or orthopaedic. Emphasis is laid throughout on everyday conditions, conservative treatment and the common operations. Where detailed discussion of controversial issues could be confusing (as on the merits of total patellectomy) a brief review of the differing ideas is given and then a personal opinion. At the end of each chapter is a list of references for those interested enough to read further; indeed, the references are almost encyclopaedic in scope. The section on quadriceps exercises, the critical review of arthroplasties of the knee and the chapter on synovial disorders are outstanding. But there are surprising omissions. Tuberculosis of the knee is not described by itself, although it is mentioned in differential diagnosis and in the section on arthrodesis; braces and splints are recommended for *genu valgum* and *varum*; but they are not described at all. The illustrations are numerous, good and apt, although occasionally they do not correspond with the text; one figure (153) does

not show an incision as described and it shows the semi-tendinous on the lateral aspect of the knee. The management of the knee in paralysis, both flaccid and spastic, is good. General diseases which affect the knee are well summarized.

DePalma avoids the common fault of describing a lot of operations without discussing their merits. In passing one must admire at least the candour of the college professor who advocates probing the knee with a finger during meniscectomy.

This book is comprehensive, up to date and readable; it should be more than adequate for the post-graduate student and the general surgeon; and if only for its bibliography it should be a valuable reference book for the orthopaedic surgeon.

Anatomy for Surgeons. Volume I: The Head and Neck. By W. Henry Hollinshead, Ph.D.; 1954. New York: Paul B. Hoeber, Incorporated, Medical Book Department of Harper and Brothers. 10½" x 7½", pp. 572, with 326 illustrations. Price: \$12.00.

This is the first of three volumes by W. Henry Hollinshead, professor of anatomy at the Mayo Clinic Foundation, Rochester, University of Minnesota. It is an unusual type of work, "not a complete descriptive anatomy, rather an attempt to describe and interpret the anatomical facts and concepts that the surgeon has found useful". The author is not a medical graduate but has had the advice of a large number of specialists at the Mayo Clinic. As this clinic is situated in a mid-western town of some 40,000 people, where there are 300 members of the clinic staff and some 500 Fellows, he has had the advantage of easy and close collaboration at all times with specialists.

Each section is complete in itself and has a very extensive bibliography. "No attempt has been made to describe the indications for the detailed technique of specific operations . . . nevertheless certain anatomical facts and concepts are of importance only as they pertain to surgery and most surgical procedures are based upon the anatomy or the physiological anatomy of the part concerned . . ."

In the section on the cranium there is an analysis of the variation at the torcular and paranasal sinuses of which, according to Woodhall, there are four types. These are the common pool, 9%, in which the straight and superior sagittal sinuses meet, and both lateral sinuses drain this channel; the plexiform type, which is provided with several channels providing adequate communication between the two sides, 56%; the ipsilateral, in which the sagittal sinus runs into one side in the straight sinuses to the opposite side, 31%; and a unilateral type, in which both the sagittal and straight sinuses enter upon the same lateral side and there is an absence of the opposite one, 4%.

Throughout there are very many references to points of neurological interest. There is reference to the work of Taarnhøj, who relieves pressure on the fifth nerve by incising Meckel's cave; and it is very interesting to note that Anderson Stuart and MacCormick are mentioned for their work on the action of the epiglottis in swallowing.

The weakest section is that on the fascia and fascial spaces. The author does not appear to have given his own views, but those which are for the most part some twenty years old. In his general description he does not seem to have realized that the fascia conforms in structure to the function of the part which it encloses, and there is no mention of more recent work which has appeared in *THE MEDICAL JOURNAL OF AUSTRALIA*.

With this exception the book can be confidently recommended to a wide variety of specialists whose practice comes within the field of the head and neck. The book is exceedingly well printed, the type is large and clear, the illustrations are wholly diagrammatic.

Diseases of the Liver, Gallbladder and Bile Ducts. By S. S. Lichtman, M.D., F.A.C.P.; Third Edition; 1953. Philadelphia: Lea and Febiger, Sydney: Angus and Robertson, Limited. In two volumes. 9½" x 6½", pp. 650, with 233 illustrations, three in colour. Price: £11 16s. 6d.

The third edition of this standard text-book on diseases of the liver and biliary system has appeared four years after the previous edition. It is now published in two volumes instead of a single one and has been thoroughly revised and brought up to date.

The preface to the third edition appears to have been hurriedly written and is disappointing. It is stated that "to provide punch biopsy for routine diagnostic use . . . would lead to its condemnation because of an increasing number of casualties", yet on page 1101 Terry, in 1952, is quoted

that among 10,600 needle biopsies the mortality was 0.12%. Again to quote: "The antibiotics are assuming increasing significance, not as near-virucidal agents but as unexplained nutritional benefactors resulting from their selective bacteriostatic action upon the intestinal flora." There is little experimental or clinical evidence to support this view.

In the summary on liver function tests and procedure a minimum selection of tests recommended for routine use is as follows: blood bilirubin, quantitative and qualitative; blood cholesterol, total and ester; alkaline phosphatase; plasma protein; total albumin-globulin fractions; prothrombin index; cephalin cholesterol flocculation; thymol turbidity; galactose tolerance and clearance; urobilinogenuria; hippuric acid synthesis and vitamin K prothrombin response. The galactose tolerance test has not been found of much value by some investigators.

The chapter on virus hepatitis is excellent and covered very fully. The importance of acute hepatitis without jaundice is stressed, the "hepatitis *sine ictero*" of Eppinger.

The chapter on hepatic coma has been rewritten and adequately reveals present ignorance in the aetiology and management.

In the chapter on hepatic cirrhosis there is an interesting historical summary of the surgical treatment of portal hypertension from Banti, who suggested splenectomy in 1898, to Learmonth, who advises splenectomy and oesophago-gastrectomy in 1952. No considered opinion is given to the reader of the relative values of the many operations listed for relief of portal hypertension.

In newgrowths of the liver there is mention of the unexplained fever of mild degree as an important criterion of diagnosis in primary carcinoma of the liver.

In the chapter on syphilitic hepatitis it is stated that "acute syphilitic hepatitis responds favourably to arsenical treatment". This statement could well be modernized in the next printing.

In the chapter on treatment in liver disease, in the section on treatment of bile stasis, the case for the use and misuse of choleretics is extremely confusing. In a mass of conflicting opinions no clear directions are given to the reader.

The author's choice of anæsthetic in operations on patients with severe liver disease is cyclopropane.

In the section on cholelithiasis it is stated that "the presence of gall-stones in the gall-bladder *per se* does not constitute a valid indication for cholecystectomy". The author also strongly recommends general medical treatment for chronic cholecystitis—the use of a daily laxative, choleretics and diet even if gall-stones are present—and states that one-third of the patients will be relieved of their symptoms. Most surgeons and also physicians would not agree with these views.

This book contains an immense amount of information on every aspect of liver disease, and each chapter is covered by a full and comprehensive list of references. For the specialist in gastro-enterology, general physician, biochemist and student it provides helpful reference and authority. It should be found on the shelves of any worthwhile medical library.

Metabolic and Toxic Diseases of the Nervous System. Proceedings of the Association for Research in Nervous and Mental Disease, December 12 and 13, 1952, New York. Edited by H. C. Merritt and C. C. Hare; 1953. Baltimore: The Williams and Wilkins Company; Sydney: Angus and Robertson Limited. 9" x 6½", pp. 616, with 104 illustrations. Price: £5 7s. 6d.

The twenty-eight papers published in this volume and the recorded discussion which follows them make up the proceedings of the thirty-second meeting of the Association for Research in Nervous and Mental Disease. They are of particular importance in that they were designed to throw light on the problem of the aetiology of the degenerative diseases of the central nervous system and the neuro-muscular apparatus. It is pointed out in the preface that it is becoming increasingly evident that the answer to the problem will be that a specific metabolic disturbance or a reaction to some toxic substance is the cause of the neuronal or neuro-muscular degeneration in most, if not all, of these diseases. Therefore, in the design of the programme for the meeting an attempt was made to have presented the known facts regarding the metabolic disturbances and toxic substances which are the cause of diseases of the nervous system, in the hope that this would lead to further studies which would give us a rational basis for prevention and treatment. The approach is comprehensive. The first group of papers deal with the relationship of the parathyroid gland, the pituitary gland, the adrenal cortex, the thyroid gland

and the thymus to diseases of the nervous system. Then the effects of excessive heat and of low environmental temperatures are considered. Two articles are concerned with the role of the vitamin B group and of folic acid, vitamin B₁₂ and related compounds in the metabolism of the nervous system. Two papers follow on abnormal copper metabolism and hepato-lenticular degeneration and on the neurological disorder associated with liver disease. Six papers deal with the relationship of lipide, carbohydrate, potassium, porphyrin, creatine and creatinine metabolism to diseases of the nervous system. Two papers relate to drugs, and two to recent physiological developments. The concluding seven papers are concerned with the action of various toxic substances on the nervous system. The contributors are distinguished, and the treatment is comprehensive. Although the volume does not pretend to solve the problem of the degenerative diseases of the nervous system, it brings together a large collection of ascertained facts and so makes an important step forward towards the elucidation of a difficult problem.

Annals of Medical Detection. By Berton Roueché; 1954. London: Victor Gollancz, Limited. 8" x 5½", pp. 226. Price: 13s. 6d.

THREE true stories, all of which have appeared in *The New Yorker*, will provide entertainment for medical and non-medical readers alike. Each story tells of an investigation into a medical problem in the United States, usually of a public health character. The first, entitled "A Pig From Jersey", deals with a sudden outbreak of trichinosis in New York and recounts how it was traced to its source; in "A Game of Wild Indians" we learn of the strange circumstances surrounding a localized epidemic of typhoid; "The Fog" describes in dramatic fashion the tragic consequences of a fog associated with air pollution in an industrial town; and so on. There are a dozen stories in all. The writing is never dull, the medical information is sound, and the author has an enviable ability to convey technical details clearly and pleasantly. While occasionally he shows the journalist's straining after effects, his work is far above the standard of the "thriller newspapers" to which he refers in one of his stories. As light reading this book can be highly commended.

The Year Book of the Eye, Ear, Nose and Throat (1953-1954 Year Book Series). The Eye, edited by Derrick Vail, B.A., M.D., D.Oph. (Oxon.), F.A.C.S., F.R.C.S. (Hon.); The Ear, Nose and Throat, edited by John R. Lindsay, M.D.; 1954. Chicago: The Year Book Publishers, Incorporated. 8" x 5½", pp. 456, with 126 illustrations. Price: \$6.00.

A WIDE range of both general and specialist literature from various parts of the world has been examined by the editors of this Year Book to obtain their material. A limited amount of critical editorial comment has been added to some of the articles. The section on the eye has chapters on the orbit and adnexa, the conjunctiva and cornea, the uvea, the lens and cataract, refraction and motility, the optic nerve, neurology and visual fields, the retina, glaucoma, surgery, therapy and miscellaneous subjects. The section on the ear has chapters on hearing and hearing tests, vestibular function and vertigo, tubal function and otitis media, otosclerosis and fenestration, facial paralysis, the parotid gland and miscellaneous subjects. In the section on the nose and throat are chapters on the nasal sinuses, the oropharynx and nasopharynx, the larynx and hypopharynx, the trachea, bronchi and oesophagus, allergy and miscellaneous subjects. While this book is primarily designed for specialists in diseases of the eye and of the ear, nose and throat, it will be useful to many others whose interests touch on these fields.

The Year Book of Orthopedics and Traumatic Surgery (1953-1954 Year Book Series). Edited by Edward L. Compere, M.D., F.A.C.S., F.I.C.S.; 1954. Chicago: The Year Book Publishers, Incorporated. 8" x 5½", pp. 362, with 269 illustrations. Price: \$6.00.

A NOTICEABLE feature of this Year Book, which will surprise no one who is aware of the overall trends of modern surgery, is the number of articles which deal with medical aspects of diseases of the skeletal system and also with basic scientific research. The articles abstracted are taken from journals received between November, 1952, and November, 1953. The abstracts are grouped into chapters on poliomyelitis, congenital deformities, the anatomy, embryology, pathology and physiology of the skeletal system, the epiphyseae, osteomyelitis and other infections, tumours, cysts and fibrodysplasia, arthritis and rheumatism, fractures and

dislocations, the spine and pelvis, the neck, shoulder and arm, the hand and wrist, the hip, leg and knee, the foot and ankle, surgical and diagnostic techniques, amputations and prostheses, instruments, appliances and bone banks, and miscellaneous subjects. The editorial comment is sparse but helpful.

The Bible and Modern Medicine: A Survey of Health and Healing in the Old and New Testaments. By A. Rendle Short, M.D., F.R.C.S.; 1953. London: The Paternoster Press. 7½" x 5", pp. 144. Price: 6s.

THE subtitle of this little book describes its contents very well. The author, who at the time of his recent death was Emeritus Professor of Surgery in the University of Bristol, is well known for his writings on surgical subjects. It is not surprising, therefore, that the present book is competently written and reasonable in its approach. After making a general survey of medical ideas in primitive times, he examines what the Old and New Testaments have to say about priests and physicians, the sanitary code, various diseases and their treatment, including leprosy, and medical folklore. Separate chapters are given to Luke the physician, the physical cause of the death of Christ, the miracles of healing, demon possession and faith healing. The book concludes with a chapter on the biblical conception of sickness. Rendle Short's attitude to the Bible is essentially reverent and conservative. While his book will appeal primarily to those who share his outlook, it has much to interest a wider circle of readers.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"A Synopsis of Obstetrics and Gynaecology", by Aleck W. Bourne, M.A., M.B., B.Ch. (Camb.), F.R.C.S. (England), F.R.C.O.G.; Eleventh Edition; 1954. Bristol: John Wright and Sons, Limited. 7½" x 5", pp. 544, with 170 text figures. Price: 25s.

Intended as a useful supplement to and not as a substitute for ordinary text-books.

"Seventy-Five Years of Medical Progress, 1878-1953", edited and with a foreword by Louis H. Bauer, M.D., F.A.C.P.; 1954. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9½" x 6½", pp. 286, with 25 illustrations. Price: 43s.

Summarizes "the story of the past seventy-five years in medicine and the present extent of our knowledge". There are twenty-six contributors.

"A Manual of Otolaryngology, Rhinology and Laryngology", by Howard Charles Ballenger, M.D., F.A.C.S., and John J. Ballenger, B.S., M.S., M.D.; Fourth Edition; 1954. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9½" x 6½", pp. 366, with 139 illustrations, three in colour. Price: 64s. 6d.

Intended for medical students, nurses, general practitioners and as a ready reference for otolaryngologists.

"The Pharmacologic Principles of Medical Practice: A Text-book on Pharmacology and Therapeutics for Medical Students, Physicians, and the Members of the Professions Allied to Medicine", by John C. Krantz, junior, and C. Jelleff Carr; Third Edition; 1954. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9½" x 6", pp. 1294, with 111 illustrations, six in colour. Price: £6 9s.

The approach is "what to do and why" and "how to do it and when".

"The Medical Annual: A Year Book of Treatment and Practitioners' Index", edited by Henry Tidy, K.B.E., M.A., M.D. (Oxon.), F.R.C.P., and R. Milnes Walker, M.S. (London), F.R.C.S.; seventy-second year; 1954. Bristol: John Wright and Sons, Limited. 8½" x 5½", pp. 596, with 99 illustrations, a few in colour.

R. Milnes Walker replaces as co-editor the late Professor A. Rendle Short.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 11, 1954.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the *Quarterly Cumulative Index Medicus*. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

TRENDS IN NURSING.

HE would be a bold man who would declare that the teaching and training of nurses were not as important as the teaching and training of doctors. We may describe nursing as the handmaid of medicine, and we recall that in the earliest times doctoring and nursing were generally carried out by the same person. The two professions have, however, grown side by side and each has had its own peculiar development, passing gradually from darkness into light and increasing its range of activities as well as its availability to mankind. Advances that are made in medicine have their inevitable result on the work of nurses, and apart altogether from what we may call the economic and social side of nursing, we can readily understand that there can be no standing still in the development of the nursing profession. As medicine advances, the profession of nursing must advance with it. During recent years, several important contributions have been made to the problems of nursing. In 1947 there appeared in Great Britain a report of a working party on the recruitment and training of nurses; this was published by the Ministry of Health and the Department of Health for Scotland. In April, 1949, we discussed in these pages a report prepared by Dr. Esther Lucile Brown for the National Nursing Council; Dr. Brown's report dealt with nursing for the future. In November, 1950, we considered at some length a report on nursing of an expert committee of the World Health Organization. All these discussions will well repay perusal even after the years that have passed. A great deal of what remained to be done in those days is still undone, and we may conclude that the subject of nursing is always, or should be always, of interest to members of the medical profession as well as to nurses. In Australia, the nursing profession labours under considerable difficulty because the period of training

required is different in the several States; for example, in Victoria a nurse may complete her training in three years, but four years are required in New South Wales. Because of the advent of the forty-hour week, the actual time devoted by a nurse to her training has been considerably shortened and, of course, the difficulties associated with that training have not become any less. Reference is made to the subject on this occasion because of the publication of an oration by Miss G. N. Burbage, Matron of the Fairfield Hospital, Victoria. The oration was delivered at a special meeting arranged by the Victorian Florence Nightingale Committee and the Royal Victorian College of Nursing.¹ Matron Burbage is concerned, and rightly, with the standards of training received by nurses in Victoria. (A great deal of what she has to say applies with equal force in other States.) She thinks that the general pattern of training today for nurses is not satisfying the patients, the members of the medical profession or the nurse herself. She reminded her audience at the outset that the function of a hospital was the care of the sick, although other activities might be carried on in the achievement of this objective. The care of the sick, the training of student doctors and the carrying out of research were not, she said, incompatible. All that was needed for the functioning of the hospital and the research programme and the training of doctors was staff to care for the patients. The problem was, and would remain, how to obtain sufficient people to do this work.

Matron Burbage quotes a statement by the World Health Organization of what is an acceptable standard for professional nurses. In spite of the length of this statement, it must be recorded here. The nurse: (a) Is prepared to recognize and to adjust to changing social, economic, medical, nursing and health situations. (b) Is well adjusted in her own living, in her work and in her relationships with others, and has developed a sense of personal and professional responsibility. (c) Has the capacity for and the will to seek continued growth and educational development. (d) Is equipped through generalized preparation to work in all fields of nursing. (e) Is prepared to give total nursing care, which includes social, physical, mental and emotional aspects. The nurse is to be trained: (a) As a nurse member of the health team, to analyse the nursing needs—physical, mental, emotional and social—of individuals, both sick and well, and to plan the nursing care necessary to meet their needs. (b) To carry out nursing technique skilfully herself. (c) To teach and supervise auxiliary workers, patients' families and community groups, appropriate nursing and health care. (d) For administration in selected situations on the wards and in the community programmes. (e) To participate in community programmes and nursing organizations. In Victoria, according to the *Nurses Act*, a number of ninety-one lecture hours is required during the three years of a nurse's training; many schools give more than this number. Overseas, the International Council of Nurses considers that about twelve hundred hours are required. Matron Burbage remarks that it may be that in Victoria nurses are doing better in the wards where they spend most of their time. She describes the ideal picture of work done in hospital wards and she adds that

¹ *Una: Nursing Journal*, May, 1954.

changes which have occurred in ward work in the last few years have produced an entirely different picture. She writes: "Some of us are watching with acute apprehension because these threaten the safety of the patient and the training of the nurses to an extent which is becoming dangerous to both. Our anxieties have been confirmed by the results of a survey in England." To Matron Burbage it is an interesting and amazing fact that the public would be terrified to let a man learning to drive take a bus full of passengers for a run, and yet gladly accepts what is virtually a schoolgirl dressed in a uniform while she learns her work, and allows her to take care of sick people, often alone. This is the faith of a public in a nurse's uniform! Basic nursing care, we are told, is left primarily in the hands of the most junior nurses—five and a half hours of the nine hours are spent in this way. A survey shows that the trained nurse, on an average, spends four and a half hours in ward organization and four and a half hours in nursing duties. In nursing duties, she has to accompany a doctor on his rounds and make contact with patients and visitors and spend time serving meals. The most disturbing aspect of the survey to Matron Burbage is the fact that in four and a half hours of organization, though the sister spends fifty minutes giving instructions to staff for the day, she spends five minutes a day teaching nurses. This means that the basic nursing is done by junior nurses who have been taught first, but who are almost entirely unsupervised in the practice of their teaching.

Matron Burbage concludes that there is great need for some organization or for a person with the well-being of the health of the community at heart to provide financial help for the research needed to delineate the Australian situation so that a nursing and health programme can be planned. What is needed is to decide what a nurse will be required to do when she is trained and then to educate her for that. At the present time, the trained nurse in hospital spends only 16% of her time on nursing and the remainder on administration for which she has not been trained. It would be useful if someone could be found to make a survey of the Australian situation in regard to the actual training of nurses carried out in hospitals. This should be possible by means of a questionnaire, and it could be done if hospital authorities would agree to cooperate.

Current Comment.

ADRENALECTOMY IN RELATION TO ADRENAL TUMOURS.

ADRENALECTOMY, especially for the removal of adrenal tumours, requires of the surgeon much more than mere technical skill in excision. The complex matters involved have been dealt with by an acknowledged master of the subject in a recent review by G. F. Cahill¹ of the various neoplasms of the adrenal gland. He includes a short description of the origins, symptoms and associated operation problems and discusses the hormonal problems involved in removal of these neoplasms. He states that stromal tumours of the adrenal are extremely rare. Medullary cell tumours are derived from the ectodermal sympathetic system and are of three types: (a) sympathio-

blastoma, from the embryonic sympathetic nerve cell; (b) neurocytoma (ganglioneuroma), from the sympathetic ganglion cell; (c) pheochromocytoma, from the pheochromocyte, a hormonal cell. Cortical cell tumours from the mesodermal glandular cells of the cortex may be adenomata or carcinomata, and may be non-hormonal or hormonal.

Sympathioblastoma, the most frequently occurring neoplasm of the medulla, is extremely malignant and occurs more frequently in infancy and childhood than later. The primary tumour is scarcely ever noticed, metastases being the first clinical manifestation. Diagnosis may involve a major hazard; handling of an adrenal mass is precarious to the patient and should be avoided completely, and the extreme blood vessel expansion of these tumours makes even perirenal gas insufflation dangerous. If removal is to be attempted in cases diagnosed early, excretion urograms are the most useful adjunct to early and safe diagnosis; they show a normally secreting kidney displaced downwards by the mass above, in contrast to the definite deformity of the calyces and renal pelvis in Wilms tumour. Likewise the surgical approach must allow wide, free and very easy removal with hardly any handling of these dangerous tumours. A wide transverse transperitoneal approach across the upper part of the abdomen is the method of choice, with early ligation of all vessels to and from the tumour. When, as is usual, metastases are present, irradiation has produced recession of the metastases; but as they recede, the tumour appears elsewhere. There are few carcinomata with a graver prognosis.

Neurocytomata are more rarely seen, and occur in a later age group. They are relatively benign, and occur in any of the sympathetic ganglia as well as the adrenal. They are well encapsulated, not very vascular and easily removed. Prognosis is good if removal is complete.

Pheochromocytomata are not frequent in occurrence, but are at present of unusual interest because of the newer steps in diagnosis and therapy. About 80% of all tumours of this type occur in the adrenal, the rest being mostly in the abdominal sympathetic ganglia. In about one-sixth of the cases the tumours are multiple. In less than 10% they are malignant. The clinical features of pheochromocytoma were referred to in these columns recently¹ and exemplified in two interesting and comprehensive case reports from Tasmania published in the same issue of the journal. It is not necessary therefore for us to follow Cahill as he covers this ground again in detail. Treatment is by surgical removal of the tumour or tumours, an operation which calls for the maximum of knowledge, experience and skill from both surgeon and anaesthetist.

Non-hormonal cortical tumours occur not infrequently. Cahill states that these are found in both sexes, more often in the fourth or fifth decades, and are silent in their early growth. They may even grow to a large size before being recognized. Pulmonary metastases occur, but some of these tumours become very large without metastasizing. With increasing size of the tumour, a mass is found on the affected side. The deep location of the adrenal gland, the slow growth of the tumour and its slowness of invasion of surrounding viscera often allow the tumour to become extremely large before discovery. Most of these tumours, though well encapsulated, are malignant. Because of the usually very large size it is often advisable to use a large incision, with a transthoracic approach through the tenth rib and then the diaphragm and extension of the incision as far down into the abdominal wall as is necessary. Because of their encapsulation these tumours are readily separated from the other fascial planes, after the vascular tree is ligated. Recovery of the patient after removal of the tumour is the rule, and freedom from metastases or recurrence is often obtained, since quite often secondary growths are not present at the time when the operation is undertaken.

Hormonal cortical tumours with symptoms of virilism occur mostly in younger patients. They occur even in children, but at this age are less common than bilateral adrenal hypertrophy. The incidence is greater in girls

¹ J. Urol., February, 1954.

² M. J. AUSTRALIA, June 26, 1954.

than in boys (four to one). Accompanying the usual virilistic changes in either sex is elevation of the 17-ketosteroid output in the urine; the amount of output varies with the duration of symptoms and size of the tumour. Cortisone is used to differentiate between bilateral hypertrophy and unilateral tumour; with the former it causes reduction of the 17-ketosteroid output, but this is not so when a neoplasm is present. There is usually no other deviation from the normal metabolism in relation to sugar, sodium or potassium levels, or to the corticoids. Once the symptoms and assays have shown the presence of an independent adrenal androgenic excretion, a tumour can most successfully be located by perirenal gas insufflation. In these cases, the fascial planes allow the gas to infiltrate easily and to outline the adrenal and a contained tumour. In practically all cases with androgenic symptoms only, and no other metabolic changes, the condition is due to benign adenoma, and cure is almost certain by operative removal. The latter is not accompanied by any risk of hormonal effects, and there is no depression of the opposite adrenal. Although the menses are resumed and hirsutism disappears, none of Cahill's patients in this group have subsequently become pregnant.

Subjects of tumours with excess corticoids show the Cushing syndrome, but tumour is less often a cause than bilateral hypertrophy. The tumours occur most often in adult females. There is rapidly developing, painful trunk obesity, with rounded "moon-face", skin striae and metabolic changes due to excess elaboration of corticoids. The corticoids may be identified in the urine, while the 17-ketosteroids are reduced in amount. Bilateral perirenal gas insufflation usually will determine the difference between bilateral hypertrophy and tumours. Angiograms are not so helpful. Failing definite localization of a tumour, it may be necessary to make a bilateral exploration. In contrast to the surgical management in the case of androgenital syndrome tumour, operative correction of an adrenal tumour associated with the corticoid syndrome involves a serious risk to the patient. Post-operative reaction is severe, and will be fatal unless steps are taken to meet the expected Addisonian crisis. The excess corticoids produced by the tumour depress the activity of the opposite adrenal; so substitution therapy must be given before, during and after operative removal of the tumour, and should be continued until the opposite gland resumes function. This takes from one to six weeks. The therapy consists of administration of cortisone and ACTH for two days before operation, then cortisone, ACTH, DOCA and salt during operation and for two days afterwards. The dosage of DOCA and cortisone is then lowered, and if no evidence of adrenal failure is present, only ACTH and saline are administered. The underlying idea is to use substitution hormones through the dangerous period, the depressed contralateral adrenal being at the same time stimulated with ACTH. These patients are "poor risks" for infection and stress. Most of the tumours are benign and not large, and are enucleated without great difficulty. Often they may be resected from normal-appearing adrenal tissue. If there is successful recovery from adrenalectomy, recession of the symptoms occurs rapidly.

Mixed syndrome tumours sometimes occur in all age groups in which the symptoms of Cushing's disease are mixed with those of virilism, but are met with more frequently in the female. Most cases have been due to rapidly growing carcinoma of the cortex. Operative procedures require the same preparation as for tumours associated with the corticoid syndrome. The incidence of malignant change is high, and metastases are common.

ABRAHAM LINCOLN: MEDICAL ASPECTS OF HIS ASSASSINATION.

In recent years, more and more attention has been paid by medical men with a flair for historical research to medical considerations affecting the lives or deaths of great men of past centuries. The latest essay in this

direction to come to our notice concerns the assassination of Abraham Lincoln, the sixteenth president of the United States of America, and is presented by Hugh R. Gilmore, junior, Curator of the Medical Museum at the Armed Forces Institute of Pathology, Washington, D.C.¹ Lincoln was shot on the evening of April 14, 1865 (six days after Lee had surrendered to General Grant) at Ford's Theatre, Washington, where with Mrs. Lincoln and two friends he was celebrating the victory of the North over the South. The assassin was John Wilkes Booth, who was the ring-leader of a plot to kill Lincoln and two others, General Grant and Vice-President Andrew Johnson. General and Mrs. Grant were to have been in the theatre party, but were obliged to leave the city beforehand; when the conspirators learned of the change, General Grant's place as the proposed second victim was taken by the Secretary of State, William H. Seward. Gilmore gives a brief account of the family history of Booth, which shows him to have been nurtured in a tradition of rebellion against authority. His father, an outstanding actor, a man of unstable temperament and a heavy drinker, was named Junius Brutus, after one of the assassins of Julius Caesar. "On one occasion, he was narrowly prevented from stabbing an acquaintance merely because he felt he had to cut someone's throat." In 1864 John Wilkes Booth had organized an unsuccessful attempt to kidnap the President. He was twenty-seven years old in 1865, a Southern sympathizer, a moderately successful actor and a plotter. The other conspirators were Lewis Paine, George A. Atzerodt and David E. Herold. Paine was allotted the task of killing Seward. He made a most determined attempt; but Seward was in bed with a fractured jaw, and his life was saved by the iron brace used to support it. Atzerodt was to assassinate Johnson, but his courage failed, and he made no attempt to do so.

Booth made careful preparations for his attack on the President. He had bored a hole in one of the two doors of the box which the party was to occupy, and between 10 and 10.30 p.m., watching through this peephole, he chose a suitable moment, entered the box, and shot Lincoln in the back of the head with a Derringer pistol (a weapon about six inches long), which fired a ball half an inch long. The bullet entered the head about an inch to the left of the mid-line, at a level which tore the left lateral (transverse) sinus. The President died at 7.20 a.m. An autopsy was performed, but the course of the bullet is still in doubt, and there has been much discussion as to how it could have entered the left side of the victim's head. The two army doctors who performed the autopsy (Assistant Surgeon J. J. Woodward and Assistant Surgeon Edward Curtis) state that the bullet lodged in the left side of the brain. But at least two other doctors who witnessed the autopsy reported that the bullet had crossed the mid-line and lodged in the right cerebral hemisphere. Inconsistencies appear also in observations on pupillary changes; Dr. Charles A. Leale, who attended Lincoln, stated that the pupil of the left eye was slightly dilated, that of the left eye was contracted, and both failed to respond to light; Dr. Charles S. Taft, who was one of the first doctors to reach Lincoln and remained with him until his death, stated that the left pupil was much contracted and the right widely dilated, and both were completely unresponsive to light. The autopsy also revealed comminuted fractures of the two orbital plates; the bullet stopped short of the frontal portion of the skull, so the fractures must have been due to an indirect force. Some authorities hold that they were examples of fractures by *contre coup*; another opinion is that they were due to a "transmitted undulatory stroke or sudden impulse of the brain substance itself against the thin bony layers constituting the orbital plates". Gilmore sums up the position by the statement that "the problem of the fractured orbital plates is the problem of skull fractures arising at a point distant to the point of impact, or as Hippocrates expressed it, 'The bone is broken in another part of the head than that in which the man received the injury'". He invites readers to decide for themselves, but himself inclines to the *contre coup* theory.

¹ *Proc. Roy. Soc. Med.*, February, 1954.

Three doctors rushed to the President's aid after the shooting—Dr. Leale and Dr. Taft, and later Dr. Albert F. A. King. The wound was located and artificial respiration was applied, since the patient was pulseless and hardly breathing. He responded a little, and about fifteen minutes after the shooting he was carried to a house across the street; it was thought that he would not survive the journey to the White House. He swallowed a little brandy, but later attempts to give it to him failed. Sinapisms were applied, and he was kept warm with blankets and hot-water bottles. Gilmore quotes the following extract from Dr. Taft's notes:

The left upper eyelid was swollen and dark from effused blood; this was observed a few minutes after his removal from the theatre. About thirty minutes after he was placed upon the bed, discoloration from effusion began in the internal canthus of the right eye, which became rapidly discolored and swollen with great protrusion of the eye. . . . The only surgical aid that could be rendered consisted in maintaining the head in such a position as to facilitate the discharge of the wound, and in keeping the orifice free from coagulum. . . . While the wound was discharging freely, the respiration was easy; but the moment the discharge was arrested from any cause, it became at once labored. It was also remarkable to observe the great difference in the character of the pulse whenever the orifice of the wound was free from coagulum and discharged freely; thus relieving, in a measure, the compression. This fact will account for the fluctuations in the pulse. . . .

About 2.00 a.m. an ordinary silver probe was introduced into the wound by the Surgeon-General. It met an obstruction about three inches from the external orifice which was decided to be the plug of bone driven in from the skull and lodged in the track of the ball. The probe passed by this obstruction, but was too short to follow the track the whole length. A long Nélaton probe was then procured and passed into the track of the wound for a distance of two inches beyond the plug of bone, when the ball was distinctly felt; passing beyond this, the fragments of the orbital plate of the left orbit were felt. The ball made no mark on the porcelain tip and was afterwards found to be of exceedingly hard lead.

Some difference of opinion existed as to the exact position of the ball, but the autopsy confirmed the correctness of the diagnosis upon first exploration.

(Gilmore points out that Taft's account states that the ball was on the right side.) It is unlikely that modern treatment could have saved the President's life; the attending doctors appear to have done all that was possible. The passing of an unsterilized probe would be regarded with disfavour nowadays, and what is even worse, Taft appears to have used a finger.

To complete the story, we have an account of what happened to the assassin. After the shooting, Booth leaped from the box to the stage and shouted: "*Sic semper tyrannis!*" In his leap he broke the lower end of his left fibula. He hobbled to the stage door, fighting off attempts to stop him, and rode off on a horse which awaited him. His subsequent adventures do not concern us here until we come to his capture and death. There is some doubt as to whether he shot himself or was shot by Sergeant Boston Corbett. Booth and Herold were captured in a barn, and as the former refused to surrender, the barn was set on fire and Corbett fired at him as he attempted to escape. Booth lived for about two hours; his death took place on April 26. The damaged vertebra and spinal cord were removed at the autopsy, and are now in the Medical Museum of the Armed Forces Institute of Pathology in Washington. Herold, Atzerodt and Paine were later tried and executed by hanging. Gilmore states that Booth had regarded himself as the avenger of the South, but that he had eliminated "the most magnanimous, the most forgiving spirit of our times—the one man who might have helped the South". There is no need to enter upon a character study of Abraham Lincoln; but we may profit-

ably recall a famous passage from his speech at his second inauguration, which took place only a few weeks before his death:

With malice toward none; with charity for all; with firmness in the right, as God gives us to see the right, let us strive on to finish the work we are in; to bind up the nation's wounds; to care for him who shall have borne the battle, and for his widow, and his orphan—to do all which may achieve and cherish a just and lasting peace among ourselves, and with all nations.

It is unnecessary to emphasize the relevance of these words at the present time; they are ageless.

HYPERTENSIVE ENCEPHALOPATHY AND ITS RELATION TO THE MALIGNANT PHASE OF HYPERTENSION.

In an editorial in *The Lancet* of June 12, 1954, recent work on the nature of hypertension was reviewed. One of the implications of the findings is that there are no grounds for regarding essential hypertension as a disease or as anything other than an arbitrary upper section of the normal range of pressures. Certainly life-expectancy decreases as the pressure rises; but the same is true for weight, and we do not feel it necessary to postulate a disease of essential obesity. One man with a high blood pressure will succumb in middle age, while another will maintain a pressure of the same order until ripe old age. The state of the vascular system rather than the height of the blood pressure seems to be of importance.

One of the conditions arising from hypertension is now called hypertensive encephalopathy. The syndrome presents itself to the clinician in many different patterns, composed of generalized convulsions, local disturbances of cerebral function and symptoms of increased intracranial tension, either singly or in various combinations. The attacks are sudden in onset and brief in duration and may occur in any form of severe hypertension. A steep additional rise in blood pressure may herald an attack. F. B. Byrom¹ has attempted to elucidate the mechanism of the production of this condition in a series of extremely fine experiments and outstanding logical deductions carried out at Saint Vincent's Hospital, Sydney. Young adult rats were used, and the hypertension was induced by excising the right kidney and, two weeks later, applying to the left renal artery a clamp with a gap of 0.01 inch. This simple procedure regularly caused severe hypertension and had the important advantage that the hypertension could be abolished simply by removing the clip from the renal artery; for the clip protects the kidney against secondary hypertensive damage.

Cerebral symptoms never occurred until the systolic blood pressure reached or exceeded a level of about 200 millimetres of mercury. Some rats developed symptoms directly this level was reached; others, with pressures ranging from 200 to 250 millimetres, persisted for as long as nine months before cerebral symptoms appeared. The parallel with man is obvious, and these rats were all presumably healthy to begin with. A steeply rising blood pressure often preceded the symptoms, as it does in man. Generalized epileptiform convulsions occurred in 34% of 250 animals. In the intervals between convulsions some of the rats appeared normal, others dazed and hyperexcitable. In other animals (44%) the presenting symptoms were weakness and apathy, deepening into coma. Other cerebral symptoms also occurred, together with vascular damage in other organs, cardiac failure and intraperitoneal hemorrhages. Death often occurred in six to forty-eight hours. Others had spontaneous remission of symptoms with relapses later. Local sympathectomy had no effect on the condition. Of the rats which recovered from the operation, removal of the clip from the renal

¹ *Lancet*, July 31, 1954.

artery, with the consequent fall of blood pressure, brought dramatic and complete removal of symptoms in all. The blood urea content of the rats was not above normal. The brains of rats with encephalopathy were examined; and while some lesions were found, such as focal arterial necroses, recent infarcts and hemorrhages, the symptoms which regularly disappear completely in a matter of hours when the hypertension is relieved, cannot be attributed to these structural lesions. Since gross oedema of the human brain is sometimes found *post mortem* in association with encephalopathy, the water content of the freshly removed rat's brain was determined. There was an increase in water content, slight at first but greater in the later stages, and this was confined to the cerebrum. In order to determine whether the oedema was general or focal, the dye, trypan blue, was injected intravenously. This dye is unable to pass the blood-brain barrier. If the capillary endothelium is damaged, the dye readily escapes and stains the brain substance. When dye was injected intravenously into normal rats and rats with simple hypertension, there was no escape of dye; but in the rats with cerebral symptoms conspicuous rounded or rosette-shaped blue spots were visible on the surface of the cerebrum. In only a few of these blue areas was histological evidence of structural damage seen. The water content of the blue spots was considerably higher than that of control areas. There are then regularly present in the cerebral cortex, in cases of encephalopathy, multiple foci of increased capillary permeability, with attendant focal oedema.

The nature of the circulatory disturbance was then investigated. That arterial spasm is a necessary incident in the production of arterial necrosis has long been believed, and this implies that the peripheral vascular resistance should be further increased in tissues or organs which show the changes noted. It was found that the peripheral resistance in the brain was increased in encephalopathy. In order to obtain direct evidence of vascular changes in the cerebral cortex an ingenious series of experiments were performed. Small windows, at first of very thin "Perspex", and later, and much better, moulded windows of transparent acrylic resin were set in the rat's skull and direct photographs obtained of the vessels with a 35-millimetre camera attached to a dissecting microscope, by electronic flashlight. In this way it was possible to record the appearance of hundreds of cerebral vessels in the normal rat, in simple hypertension, in encephalopathy and, finally, after abolishing the hypertension. In simple hypertension the cerebral arteries remained normal in appearance or occasionally showed slight diffuse narrowing. In encephalopathy the changes were well marked and seen regularly. In the smaller cerebral arteries the typical change consisted of conspicuous uniform narrowing, often intense enough to make the affected branch almost invisible. The change, though widespread, was usually patchy. In the larger arteries the changes were more localized. Two weeks after abolition of the hypertension the arteries appeared to be normal.

By a careful examination of his material Byrom concluded that other factors, such as strain due to oedema, could not be the cause of the vessel changes; only arterial spasm was left, and he holds that this is a direct response to the increased intraarterial pressure. This postulate finds sound physiological support in a paper by Bayliss, published fifty-two years ago, in which he showed that arterial muscle has an intrinsic tone which is governed largely by the intraarterial tension. The normal artery contracts against a rising pressure and relaxes when the pressure falls. Bayliss demonstrated that the reaction was local and myogenic. Arteries in the intestinal wall were also shown by Byrom to exhibit intense local spasm in rats with encephalopathy.

It seems fair to conclude that the results of direct observation lend strong support to the view that focal arterial spasm is at the root of hypertensive encephalopathy and arterial necrosis. The evidence adduced indicates that, in the hypertensive rat, encephalopathy and focal arterial necrosis, which form the essential lesion of

malignant hypertension, are different expressions of a common local circulatory disturbance, which can be described in some detail and related to a simple physiological property of arterial and other plain muscle. If intraarterial tension is both a normal stimulus to contraction and the direct cause of the spasm of encephalopathy and arterial necrosis, it is difficult to avoid the inference that it must contribute directly and progressively to the vasoconstriction of the intervening hypertensive state. It may be that the initial vasoconstriction represents the response of abnormally irritable arteries to the physical stimulus of a normal blood pressure. Although the symptoms displayed by the rat are almost exclusively cerebral, the underlying disturbance is more widespread. Focal arterial necrosis, with or without local oedema, is commonly found in the heart, pancreas, intestine and other organs, and focal spasm is regularly seen in the intestinal arteries. The arterial spasm can be promptly relieved by lowering the blood pressure. The relation of these findings to various clinical types of encephalopathy and cerebral oedema is discussed in some detail by Byrom, and the suggestion is made that the different types are different expressions of this common morbid process.

MEN, WOMEN AND TYPES OF MOTOR-CAR ACCIDENTS.

AN interesting sidelight appears in a recent bulletin (Bulletin Number 23 of 1954) issued by S. E. Solomon, the Government Statistician, Brisbane, on the subject of road traffic accidents in Queensland. A survey has been made of accidents involving only motor-cars in 1952-1953 from the point of view of whether the drivers were men or women. It was found that in collisions between cars, for every 10,000 men drivers 846 women drivers were involved as the parties not to blame. Similarly, in collisions with pedestrians which were considered to be due to pedestrian faults, the ratio of women to men drivers was 845 to 10,000. From this it may reasonably be concluded that women drove approximately 85 miles for 1000 miles driven by men. On the other hand, the drivers held to be at fault in motor-car collisions included 92 women for every 1000 men. Similarly, drivers in accidents involving one motor-car only (for which the fault must therefore attach chiefly to the driver) were in the ratio of 94 women to every 1000 men. It appears, therefore, that the frequency with which women drivers were involved in accidents was a little greater than in proportion to the amount of driving performed by them. Their excess liability over that of men was in the vicinity of 10%.

If, therefore, we assume that the probability of being responsible for an accident was 100 for a male driver as compared with 110 for a female driver, an interesting comparison can be made of the types of faults for which drivers of each sex were responsible in 1952-1953. In the first group the following faults are listed: "following too closely, excessive speed, dazzled by headlights, swerving or stopping suddenly, defective steering"; the proportion of those involved was 21 men to 10 women. In the second group the list is as follows: "failing to give proper signals, reversing without care, inattentive, not giving right of way, inexperience"; here the proportion was 31 men to 49 women. In the final group, which covers all other faults, the proportion was 48 men to 51 women. Thus, Mr. Solomon comments, in the first group of faults, which seem generally to have involved over-speedy or rash driving, men committed errors more than twice as often as women. In the second group, which includes most of the faults of careless and inattentive driving and was more important for both sexes, women were more prominent than men. The remaining faults were shared fairly evenly by men and women. These findings provoke interesting reflections, but everyone will not draw the same conclusions. Certainly, it would be a rash man (or woman) who generalized on the psychology of the sexes from a single statistical observation.

Abstracts from Medical Literature.

DERMATOLOGY.

Topically Applied Oestrogen Cream and Acne Vulgaris.

H. H. SAWICKY, J. L. DANTO AND W. S. MADDEN (*Arch. Dermat. & Syph.*, July, 1953) set out to evaluate further the clinical efficiency of topical applications of oestrogen-containing creams in the treatment of *acne vulgaris*. On one side of the face approximately one gramme of cream base containing 2.5 grammes of water-soluble conjugated oestrogen substance (equine) was rubbed in once a day at bedtime. On the other side of the face the oestrogen-free cream base was applied in like manner. On arising next morning, the patient washed his face with ordinary soap, and no other treatment was given. The patient did not know which was the active and which was the control cream. Twenty-six patients (17 female, 9 male) were followed for from two to sixteen weeks. The total amount of hormone applied on one side of the face ranged from 38 to 204 milligrammes. The three patients who showed the greatest improvement on the side treated with the hormone were male patients with numerous large cystic lesions. Patients whose predominant acne lesions were papules and pustules showed no discernible improvement.

Etiology of Nummular Eczema.

L. P. FOWLE AND J. W. RICE (*Arch. Dermat. & Syph.*, July, 1953) state that nummular eczema occurs in sharply defined coin-sized vesicular plaques favouring the extensor surfaces of the extremities below the knees and the elbows. The authors' study was based on 600 patients with an eruption on their hands; 178 patients with nummular eczema were compared statistically with those having other types of dermatitis. The authors consider that the causes of nummular eczema are multiple and that the clinical picture is dependent on the interaction of three aetiological factors, which they conceive to be nutritional, allergic and infective. In the series of patients with nummular eczema, the female patients slightly outnumbered the male. Most of the patients were in the third decade of life. It is the authors' belief that the plaques of nummular eczema represent islands of sensitized skin which mostly result from allergy to the endotoxins of the staphylococcus, sometimes to streptococci and less frequently to food proteins. The incidence of tonsil infection was significantly high in patients with nummular eczema as compared with other groups. The authors relate the importance of foci of infections to the allergic component, and believe that in nummular eczema the foci release allergens which activate the plaques of sensitized skin. They found that the incidence of staphylococcus infections, such as styes, furuncles, circinate impetigo and osteomyelitis, was significantly higher with nummular eczema than with other eczematous diseases. The incidence of pathogenic forms of staphylococci is significantly high in plaques of nummular eczema. "Vioform", which works well in the treatment of nummular eczema, has a

well-known staphylococcidial action. The use of tars reduces the number of colonies of pathogenic staphylococci in the eczematous plaques. The antibiotics, provided the staphylococci are sensitive to them, are good remedies for the control of the infective element. The eruption in many patients with nummular eczema clearly begins as *dermatitis venenata* or allergic contact dermatitis. Those patients whose nummular eczema develops in the wake of a compensable allergic eczematous contact-type dermatitis often cause embarrassment for the attending physician and the compensation adjuster. The course of the eczema may be prolonged beyond all reason when considered as an allergic eczematous contact-type dermatitis, and acute flare-ups may occur while the patient is at home away from all industrial contacts. Nummular eczema as a disease entity can be satisfactorily explained by the triad of aetiological components: poor nutrition, bacterial allergy to the allergens of the staphylococci and bacterial invasion of the skin by the staphylococcus.

Treatment of Pustular Psoriasis and Pustular Bacterid with Quinacrine.

F. E. CORMIA AND M. H. NOUN (*Arch. Dermat. & Syph.*, September, 1953) state that three patients with pustular psoriasis occurring in conjunction with psoriasis elsewhere on the body were treated with quinacrine hydrochloride. All the pustular lesions underwent involution within a three-weeks interval. One of the patients with pustular psoriasis had extensive inveterate treatment-psoriasis, with lesions scattered over the body; these scattered lesions underwent 95% involution after three weeks of quinacrine therapy. Two patients with pustular bacterid were treated with quinacrine; all the pustular lesions disappeared within a three-weeks interval. The lesions of pustular psoriasis reappeared promptly in one patient after temporary interruption of therapy; the pustular lesions again disappeared after reinstitution of quinacrine therapy.

Intractable Verruca Plantaris.

H. L. DU VRAY (*J.A.M.A.*, July 25, 1953) states that *verruca plantaris* and deep-seated calluses occur commonly under the metatarsal heads and often become a major disabling problem. Prevailing treatment often leaves a disabling residual scar. Formation of this type of newgrowth occurs most frequently under the middle three metatarsal heads. It is open to question whether many of these growths are verrucae. The author sees many seed-like calluses under the plantar condyle surfaces of the metatarsal heads containing white connective tissue fibres of a reactive fibrosis. Such a callus is often diagnosed as *verruca plantaris* and treated by irradiation, escharotics, electrodecastration or surgical excision. The mass of scar tissue resulting has been greater than the original excrescence with increased disability. The plantar surface of the head of each metatarsal has two condyle projections. Those of the middle three metatarsals end in sharp points extending proximally. The condyle on the fibular side is always the larger. The condyles during walking bear an added pivotal backward thrust for a short period each step. The thrust

results in the condylar points gouging into the soft understructures. This occurs especially with depression of the anterior arch. The skin under such condyles accumulates horny cells as a protective measure. The author advises excision of the plantar condyles with intractable growths immediately under the head concerned. The operation is described.

Chondrodermatitis Nodularis Chronica Helicis.

V. D. NEWCOMER, C. G. STEFFEN, T. H. STERNBERG AND L. LICHTENSTEIN (*Arch. Dermat. & Syph.*, September, 1953) survey 94 cases of *chondrodermatitis nodularis chronica helicis*. They state that the condition is manifested by a small painful nodule on the ear having a distinctive pathological picture. The helix is usually the site of involvement, but the condition can occur in the antihelix. The lesions are red and usually have a firmly attached centrally located crust, the removal of which reveals a small well-defined ulcer with an erythematous base. Episodes of inflammation and drainage of a purulent exudate from beneath the crust are often observed. The patient experiences intense pain at such times, particularly when he lies on the involved ear. The condition is more prevalent amongst males. Trauma of various types has been considered to be the primary aetiological factor. Eichenlaub elicited a history of frostbite in half of a series of 26 cases. Many forms of treatment have been used including X-ray and radium therapy, application of carbon dioxide snow, application of trichloroacetic acid, desiccation, and curettage and excision. Clinical reports would indicate that wide excision including the involved portion of the cartilage gives the most satisfactory results. Occupation does not appear to play a part. The majority of patients were older persons. Recurrence is not infrequent even after excision, being more common in the first year after excision. With procaine injections (2% solution given subcutaneously into and around the nodule daily for seven days) seven patients were temporarily cured. Local application of bacitracin ointment, as suggested by Nelson, was also tried in eight cases; three patients experienced subjective improvement. The authors studied biopsy specimens, and these indicated that the painful nodule on the helix represents essentially a vascularized organizing inflammatory focus within the cutis. The skin, being relatively thin at this site and tautly stretched over the cartilage of the ear, is vulnerable to minor trauma, particularly when senile changes are present. The inflammatory nodule, once established, commonly extends to the perichondrium, but the auricular cartilage itself shows only degenerative changes incidental to aging. The best results from treatment were in cases in which the nodules were injected locally with 0.5 to 1.0 millilitre of a solution containing 2% procaine solution and a 1:1000 solution of histamine sulphate.

Unusual Forms of Solar Dermatitis.

R. J. MORGAN, P. O. SHACKELFORD AND J. H. LAMB (*Arch. Dermat. & Syph.*, April, 1953) adopt the following classification of solar dermatitis based on types: (i) plaque-like type, (ii) contact eczematous type, (iii) papular-like and prurigo-like types, (iv) erythematous

type—*erythema solare perstans*. They report a series of patients. The first had vesiculo-bullous and prurigo-like lesions with scarring of a peculiar yellowish-brown colour and a leathery texture. He did not have porphyria, but had cirrhosis of the liver. The authors state that the lesions of the second and third patients are not readily placed in the classifications listed. They probably should be described as pseudocolloid milium or verrucose solar dermatitis. So far as the skin is concerned, the activity of steroids in maintaining the connective tissue and ground substance is of great importance. When androgen activity is depressed, the production of melanin is depressed. With low levels of androgens in the skin, incomplete enzymal reactions in the tyrosine-melanin system may result in the formation of intermediate products and not mature (black) melanin. Because of the lack of black melanin in the basal cells of the epidermis, strong sunlight is not filtered from the corium. Thus the dermal vessels and the collagen are subjected to abnormal amounts of actinic radiation with resultant vesiculation, prurigo induration and verrucoid and plaque-like eruptions of solar dermatitis. In the presence of deranged liver function, profound changes in steroid inactivation may be anticipated.

UROLOGY.

Retroperitoneal Teratoma.

R. CHUTE, S. E. LEARD AND R. OSGOOD (*J. Urol.*, September, 1953) report a case of the rare malignant condition of retroperitoneal teratoma. They state that symptoms are the result of pressure on neighbouring structures by the growing neoplasm. Developed symptoms are abdominal or back pain, often accompanied by symptoms of partial intestinal obstruction, and by deterioration in the general condition. In most cases a mass is discovered on physical examination of the patient, although no mass was detected pre-operatively in the case reported. In this case, excretion urography was found of real value in the diagnosis. A cystic mass, somewhat larger than a golf ball, lay behind and medial to the kidney, and therefore displaced it laterally and anteriorly. Pressure on the ureter and pelvis caused poor emptying from the kidney, which was seen to be hydronephrotic. It was found that the cystic mass was at the upper end of a firm, fixed mass resembling a large-sized cucumber; this ran down to the bifurcation of the aorta, which it covered. The mass was considered inoperable, so biopsy was taken, and this showed the mixed characters of a primary malignant teratoma. Deep X-ray therapy was given over the next two months, and the patient did well for four months; but recurrence followed, with death nine months after operation.

Wilms Tumour in Children.

R. A. GARRETT AND H. O. MERTZ (*J. Urol.*, November, 1953) report a series of 24 cases of Wilms tumour in children. They state that success in treatment is possible only with early diagnosis and rapid treatment. Nephrectomy should be carried out as early as possible, but it may be necessary to use irradiation pre-operatively in order

to reduce the mass to operable size. Should the tumour remain large in spite of irradiation, the operation must be performed transperitoneally or transthoracically. These routes allow early pedicle ligation and afford access to abdominal metastases. Finally, routine post-operative irradiation at the nephrectomy site and to any metastatic area is highly valuable. In the series of cases under study, a two-year survival rate was realized in 42.9%.

Hæmangioma of the Renal Pelvis.

J. B. ANDERSON *et alii* (*J. Urol.*, December, 1953) state that renal hæmangioma is a rare benign tumour. It normally occurs in patients under forty years of age. It may occur with or without pain. The bleeding which usually occurs may be persistent or intermittent, slight or profuse, and may be present for from a few days to as long as thirty-six years. Diagnosis is only rarely possible before the kidney is removed. The pyelographic changes may be slight or even absent, or they may simulate various other diseases. The disease is unilateral, and the lesion may vary in size from a pinpoint to a mass occupying one-third of the renal mass. The lesion is considered a true neoplasm rather than just a dilatation of existing blood vessels. Attempts to diagnose the cause of unilateral renal bleeding observed cystoscopically must be made by repeated retrograde pyelography. Although only 70 cases of renal hæmangioma have been reported, it is likely that this disease is more common than this total indicates. Since the disease is unilateral, nephrectomy has been the common treatment.

Vesical Neck Contracture in Children.

H. A. GAILLEY AND J. W. BEST (*J. Urol.*, December, 1953) state that in recent years doubt has been cast on congenital valves as the cause of obstruction in the posterior urethral and bladder-neck regions, and their own small experience is that the lesion is usually a contracture of the region of the internal meatus. They give details of two recent cases in which the internal meatus was found to be sclerotic and tightly stenosed. Infection and back-pressure effects on the upper part of the urinary tract were treated with an indwelling catheter and administration of antibiotics, and then the disease was treated by transvesical (suprapubic) approach with dilatation of the internal meatus and excision of fibroid tissue. Cystostomy drainage was maintained for ten days after each operation. Although in many recent reports the perurethral (endoscopic) approach is advised, the authors prefer the open, transvesical method, and the results so far have been gratifying.

Cystoscopic Manipulation of Ureteric Calculi.

J. H. IWANO AND R. C. BUNTS (*J. Urol.*, November, 1953) give the detailed histories of nine cases in which complications, more or less serious, arose from the attempt to remove calculi from the ureter by "stone baskets" and other such mechanical appliances. They consider that such complications occur all too frequently and state that gentleness is an absolute requisite in manipulative treatment of ureteric calculi. All instruments should be routinely checked

just before use in case a filiform guide or tip becomes detached and left in the ureter, as is described in one case. "Stone baskets" should not be used for calculi in the upper half of the ureter, or if the calculi have a greater diameter than 0.5 centimetre. In most cases it is wise to leave a ureteric catheter in place for twenty-four to forty-eight hours after any special instrumental manipulation, and antibiotics should be given before and after manipulation. Some of the complications were so severe (for example, rupture of the ureter) that the intended conservative procedure became a formidable radical one.

Vesical Carcinoma.

J. J. CORDONNIER (*J. Urol.*, February, 1954) states that the most controversial subject in urology in recent years has been the role of total cystectomy with uretero-colonic anastomosis for carcinoma of the urinary bladder. When such authors as Colby and Jewett condemn the procedure as almost valueless, it seems that the relevant data should be studied, so that the true place of this radical procedure can be evaluated. One of the reasons for poor results is that most urologists reserve this procedure for advanced and very malignant types of carcinoma. With the advent of improved methods of uretero-sigmoid anastomosis and modern chemotherapeutic agents, cystectomy can be recommended at an earlier stage, with consequent reduction in the morbidity and mortality of the procedure. Some of the problems to be overcome in evaluating types of tumours and deciding on types of operation or management are: (i) multicentric foci of epithelial change throughout the vesical mucosa, (ii) varying degrees of malignant change in different parts of the same tumour, and (iii) changes in the degree of malignant change of bladder carcinoma under continuous observation. All grade 1 lesions (mucosal only) are amenable to endoscopic resection or to electrocoagulation. In grade 3 lesions (penetration to more than half-way through the muscle) and in grade 4 lesions (right through the bladder wall) the outlook is hopeless. It is in the large group of grade 2 lesions (penetration of the muscle to less than half its thickness) that the greatest opportunity for advancement lies. When such lesions exist, early cystectomy should be advised, and accepted by the patient, as the only hope for cure. The author has reported a series of 69 cases. At the end of one year, six patients having been operated upon, all were alive. In the one to two years group, five were alive and three dead. In the two to three years group, seven were alive and six dead. In the three to four years group, seven were alive and ten dead. In the four to five years group, ten were alive and fifteen dead. The average survival figures were as follows: grade 2 growths, ten patients, average survival seventeen months; grade 3 growths, seven patients, five months; grade 4 growths, nine patients, nine months. The figures concern grading by type of cell. Survival average time in groups based on degree of invasion of the bladder wall were: (i) mucosal and superficial muscle penetration, six patients, thirteen months; (ii) deep muscle penetration, five patients, five months; (iii) right through vesical wall, three patients, eight months.

On The Periphery.

AN ACCOUNT OF STATE OF HEALTH AND PROBLEMS OF MEDICAL INTEREST IN GOILALA, PAPUA.

Historical Survey.

A GOVERNMENT STATION has been organized at Tapini in the centre of Gollala, Papua, for but a few years. Previously the only contact with Europeans had been with patrols which moved quickly through the area, being on numerous occasions forced to retreat before hordes of inimical savages. Many of these early expeditions were undertaken as punitive patrols following reports of raidings and murders in inter-village warfare. Missions have been working in Gollala from scattered stations for some thirty years; their main intent has been the establishment of buildings and communications. Thus it will be appreciated that the indigenous people have had very little contact with Europeans. Since World War II a more intensive effort has been made to maintain a permanent government station with district services and medical service. However, in 1952 patrol through the area still reveals the persistence of what is essentially a community at a primitive stage of evolution. This may be correlated with the nature of the country.

Topographical Features.

Gollala is delimited approximately by longitude 146° 37' to 147° 30' east and latitude 8° 00' to 8° 45' south. The estimated area is said to be 3000 square miles. The district consists of some eight very deep, narrow river valleys, of which Tepala, Kunimapa, Ivani, Iballa, St. Joseph and Dilava are the most important. Separating these rivers and following their intricately tortuous courses are mountain ranges rising, on an average, to between 5000 and 7000 feet. Alluvial flats are completely non-existent, as streams rush swiftly down precipitous courses between almost vertical banks up to 150 feet in height. Above the bed of the river the mountains rise acutely to very sharp summits, often no more than a few feet in transverse extent. Jagged outlines not yet weathered to less abrupt inclines give a general impression of a geologically young country.

Climate.

The rainfall approaches 100 inches annually, and rain falls mainly in December, January, February and March. Since all the villages are above 4000 feet, the climate cannot be regarded as tropical in the generally accepted sense of the word. The summer is equable. The nights are cool to cold. Rain falls in the afternoon and evening; the mornings are fine and sunny.

Government Station.

Tapini is built on a small ledge some 50 acres in extent, at an altitude of 3250 feet, 1500 feet above the course of the Iballa river. Here is the base hospital for medical service in Gollala. Hospital and wards are constructed of native materials—thatched grass roofs, rough-hewn floors and walls of most unsubstantial water-proofed paper. It is almost impossible to keep pace with natural decay and the ravages of boring ants. The procurement of European building materials is a practical impossibility. However, owing to the persistence and personal effort of the medical officer during 1951, a new hospital building is well under construction. This will consist of an operating theatre, surgery, office, dressing room and bulk store. It is hoped to procure more equipment when the new hospital is completed.

Surgical instruments are at a minimum. Many have been added from the personal equipment of the medical officer. The operating theatre is small, with an operating table of the portable army type, an instrument table and cabinets. Shutters of wooden frames and paper must be left open during the day to allow light sufficient for working. On dull days lighting is deplorably weak. After a few days' fine weather, the grass-thatched roofs leak abundantly into the operating theatre when rain falls. On dry days dust from the work of borers settles to the theatre floor. Male and female wards are of similar structure to the present hospital—rough hammocks have been made of sewn bags. Patients prefer to sleep on the floor crowded about smoking fires, to which they are accustomed in their villages. Two to three blankets are issued to each patient on arrival at the hospital. In the future new wards will be built and wooden platforms built for sleeping.

It is impossible to realize the infinite difficulty of building permanent structures without seeing how materials have to be acquired. Added to difficulties experienced all over the world by contractors—shortages and increased costs—there is here the difficulty of transport. A light aircraft of which the maximum load is 1400 pounds visits Tapini. The aerodrome is precarious; it is on a steep incline and abuts almost on a cliff 1500 feet high. It is impossible to bring timber to the station by air. All building framework is hand-sawn by native pit-sawyers from locally grown pine. Preparation of timber of the exact dimensions required necessitates patience and much labour.

Medical Staff.

The staff consists of one European medical officer, two senior native medical boys who speak English and two native medical orderlies who speak only Motu; the last-mentioned are of use only for mechanical dressings. A population of 26,000 people is being given a medical service by one doctor, who is expected to control an area of 3000 square miles and almost 100 miles across. At intervals, for short periods, a European medical assistant is stationed here, and during these times medical patrols can be executed. In the absence of the European medical assistant, the medical officer must make forced patrols to investigate reports of epidemic disease.

Since a permanent government station has been established for only seventeen years, and that period has been interrupted by the dislocation due to the war, it is not surprising that there are few roads in this area of 3000 square miles of rugged mountains and torrential rains. Such roads as have been built are one and a half fathoms wide and built at gradients not greater than one in seven. However, landslides, rocky ledges and constant overgrowth with brush make these "roads" almost impassable save by walking in single file. Apart from these government-directed native-built roads, the district is traversed by innumerable native paths, whose chief and only blessing is that they usually form the shortest route between the top of the mountain and the river-bed. Broken by unnumbered roots, often ascending almost vertically for hundreds of feet at a time—more like ladders with footholds of roots of trees, slippery traps for the unwary in wet weather—they are arduous climbing at all times. On these tracks it is everyday practice on patrol to climb 1200 feet per hour for three hours and then descend equally on the other side. All equipment must be carried on the backs of native carriers. Indeed, they exhibit remarkable stamina in bearing all day loads of 30 pounds each, while the European exerts himself to walk unhindered. Twenty to thirty carriers are employed to bear patrol equipment, food and medical supplies.

Patrol.

On routine patrol, it is customary to take antimalarial drugs, sulphonamides, penicillin, "Salvarsan", bismuth, sulphur, adrenaline, "Coramine", dressing materials, anti-septic and a few minor surgical instruments. With the foregoing equipment it is possible to treat most illnesses in villages; more prolonged and detailed treatment is undertaken after the dispatch of patients to hospital. This often means that a sick person must travel up to 70 miles—three days' walking—before hospital is reached. Since they know little of the value of medicine, their reluctance to go to hospital is well understood. The patient is accompanied by the village policeman and adult friends. Fear of passing alone through enemy country is strong. Medical patrol gives ample opportunity to study the people in their own environment, largely uninfluenced by European thought or attempts at modification.

The central theme of the organization of the life of the native is the procurement of adequate supplies of food. When it is considered that, by Australian standards, practically no conveniently arable land exists, the achievement of cultivation is remarkable. The natural forest contributes but an insignificant moiety to the diet. All gardens must be cleared and planted on steep slopes. The soil no doubt is fertile. The considerable problem of water erosion is countered in a number of ingenious ways. A garden cleared and planted customarily bears fruit for one year only, and then a new site is chosen and planted. Thus no excessive drain is placed upon the natural fertility of the soil. After ten years, secondary growth of old gardens is cleared and they are planted again. This system of rotation of gardens is prevalent throughout the territory where abundant land is available.

Most of the heavy timber-felling and burning of the larger trees is carried out by the village's males. Several

hamlets or families combine their efforts in one garden. Fences to keep out wild and domestic pigs are strongly built of stakes of suitable size selected from the timber felled. Most of the trunks not burnt are distributed horizontally across the slope of the hill; many trees on steeper slopes are merely ringbarked and left standing; these two practices limit soil erosion. The men, having completed the foregoing work, take little part in the subsequent cultivation of crops.

The women and the older girls plant sweet potatoes, pumpkins, cucumbers, taro—a valuable root crop—yams, maize, tapioca, tomatoes, English potatoes, tobacco and bananas. Extensive digging of the whole garden is not practised. Seeds or roots of various plants are sown in holes dug by sticks. Seldom is there any lack of rainfall, and from the time of planting almost all crops grow with little attention. The women attend to any necessary weeding during the period of growth. In Gollala the above-mentioned crops mature in succession, so that with some overlapping on either side the village may be living on one type of product—for example, sweet potato—for several months, quite unrelieved by any supplement.

Food.

Gardens provide the bulk of the food for all the natives. The diet is almost exclusively carbohydrate, as is seen from the crops planted. Seasonal supplements of pandanus and okari nuts supply rich sources of oils and to a certain extent vegetable protein. In some villages pumpkin leaves are used as green vegetables. Sugar-cane is grown in the warmer valleys. A few villages have orange trees planted by missions. Protein is a rarity; birds, opossums and pigs are its only sources. Pigs are kept by every family group, and indeed a man's influence is estimated by the size of his herd of swine. However, the killing and eating of pigs are limited by very strong traditional bonds. Only at funeral or dance feasts are pigs slaughtered. Then the villagers, ravenously hungry for animal protein, eat hugely of the animals. Custom dictates that no man shall eat of the pig he himself has bred. His neighbour's pig is killed for him, and in his turn the neighbour eats his friend's pig. Unwritten laws fix the rules of equity of exchange.

Although this monotonous diet of carbohydrate is thus occasionally supplemented with protein, it might be conjectured that amino acid and vitamin deficiency would be common. However, little evidence of malnutrition was observed among the natives directly examined in January, 1952, except in one local incident, where the gardens had been exhausted by a dance. According to the "Report of New Guinea Nutrition Survey Expedition" (1947), made in five villages scattered through Papua and New Guinea, absolute deficiency was found rarely. The caloric intake (1600 Calories) was below the comparable figure (2570 Calories) taken from a survey in Sydney (1944). Sufficient food was available to satisfy appetites in most villages, so it must be presumed that caloric intake is adequate. The average protein intake, from the above-mentioned report, was found to be low by European standards (New Guinea 19 to 41 grammes per day, Sydney 93 grammes). In Gollala, as in the villages covered by the survey, custom allows adult males the greatest proportion of protein available, so that children in the years of growth suffer most severely from the low protein intake. The content of vegetable proteins of the various roots and leaves eaten has not been investigated. From this source natives may acquire a considerable proportion of protein requirements. The fat intake is low, animal fat and seasonal supplements of vegetable fat from nuts being the only sources. As for vitamin and mineral intake, no accurate quantitative survey has been made in this district, nor was it possible on the patrol reported. Nevertheless, figures from the report of the Survey Expedition may be quoted as indicative of the probable intake (Table I).

The high thiamin content prompted an analysis of the various foods, of which the following are examples: yam, 0.94 milligramme per 1000 Calories; taro, 0.69 milligramme per 1000 Calories; banana, 0.35 milligramme per 1000 Calories; white bread, 0.39 milligramme per 1000 Calories. No signs of vitamin D or thiamin deficiency were seen.

Additional to the foregoing general remarks about the diets of these people, it may be mentioned that two cases of clinically diagnosed hepatic cirrhosis were observed. Both the subjects, one female and one male, had physical signs of "hepatic facies", emaciation, enlarged, hard, knobby liver, peripheral oedema and ascites. The female patient confirmed the diagnosis with an observed haematemesis. Speculation may be raised as to the etiology of this cirrhosis.

Cirrhosis following hepatitis and cirrhosis due to nutritional deficiency may be considered. In view of what seems an obvious protein and hence amino acid deficiency, the latter seems the more probable. Though this is merely an indication, further investigation may divulge a greater incidence than is at present observed.

Salt intake as sodium chloride (free) is almost nil. During the last twelve months these natives have not had any supplies of government salt. As a consequence a physiological adjustment in diet has been made. The consumption of water is very low. Many of the meals are completely without fluid. Pumpkin, taro, sweet potato and corn are all eaten baked dry in the ashes of the fire. Though it is conceded that combined water in food probably accounts

TABLE I.

Substance.	Mountain Village.	Sydney.
Calcium	0.6 gramme per day.	0.8 gramme per day.
Thiamin	0.7 milligramme per 1000 Calories.	0.41 milligramme per 1000 Calories.
Ascorbic acid	185.0 milligrammes per 1000 Calories.	—

for much of the essential daily intake, the total is well below European standards. A local source of mineral calcium derives from the habit of eating burnt lime with betel nut. Iodine deficiency—indirectly deduced from the endemicity of goitre—was very localized.

Housing and Villages.

As has been stated previously, the people live in small hamlets, groups of from three to ten houses, which cannot be called villages when comparison is made with the larger coastal settlements. Hamlets are widely scattered—one hamlet to every major spur (*lavava*) that descends from mountain tops to river valleys. On subsidiary spurs close to the hamlet are gardens; in the surrounding bush the village pigs roam and feed. Houses are built on each side of a central cleared space. Most villages are fenced to keep out wild pigs. The huts average 15 feet by 12 feet in size; the height varies. All are constructed of native materials, according to what is most abundant locally. In a few areas huts are raised two feet above the ground so that pigs may live underneath. However, in the majority of hamlets the pigs are brought into the women's house at night and there closeted off in small palisades of stakes driven into the earthen floors.

There is always but a single entrance measuring two feet by one foot, and inside a ceiling on which food is stored. On either side of the central fireplace most houses have cane or wooden platforms some nine inches above ground. Very seldom do natives actually sleep on the ground in permanent huts. The fire burns all day and at night is stirred, and soon this almost completely closed shelter is filled with warm air and much smoke. Being born, living and dying close to these fires, for whose smoke there is no exit save by slow filtering through the interstices of the closely thatched roofs, the inhabitants suffer from chronic dry coughs, and it is said that pulmonary radiography reveals greatly increased bronchial marking according to European standards. Food is cooked on the central hearth, and warmth from the fire is the only protection of these people from cold. Sudden exit from these warm, closed atmospheres to the rainy, windy and cold outside elements may have much connexion with the vulnerability of these people to pneumonic complications of influenza. Houses for males are always of higher standard than those for females. Females, young children and pigs live together crowded in very low-roofed houses, and often without the cane platforms built above the fire.

Pigs are regarded in the light of valuable domestic pets. Two years ago it was still a custom for women to suckle piglets to the exclusion of their own infants. In wet weather their wallowing close to the dwellings produces most unhygienic surroundings. Very little evidence is seen in any village of vegetable rubbish. The cleanliness of the villages varies greatly. In no case was there seen to be adequate disposal of faeces at a sufficient distance from the village to eliminate the fly-breeding menace. Sanitary standards are primitive; personal washing is unknown.

Pattern of Disease.

There follows later a statistical report of the pattern of disease. Here it may be timely to record the general trend as seen both on patrol and also through examination of admissions to the hospital. Absence of many diseases common in European communities is real in part only. Diagnostic methods and opportunities for examination have been and still are at fault in many cases. When greater staff and material facilities are available a more detailed picture will reveal many diseases at present absent from the pattern. It must be remembered that the high incidence of malaria in the following summary of six months' admissions to the hospital is drawn from Tapini base, where *Anopheles punctulatus punctulatus* has been found breeding in small, sunny, temporary pools within 200 yards of the hospital. The admissions to Tapini hospital from January, 1951, to June, 1951, were as follows: malaria, 36 patients; lymphopathia venereum, 28; frambesia, 24; tropical ulcer, 10; influenza, nine; trauma, seven; pneumonia, three, upper respiratory tract infections, impetigo contagiosa, scabies, gonorrhoea, fractures and dysentery, 28. The observation of the medical officer that the incidence of malaria has decreased in the current wet season may be due to two factors: (a) the development of immunity to plasmodia in a previously non-immune population; (b) more efficient malarial prophylaxis—the daily administration of 200 milligrammes of "Paludrine" or mepacrine. On medical patrol, as is recorded below, yaws, tinea and scabies form the commonest conditions seen. Small traumatic ulcers were very common.

In one localized division of a group of hamlets, that of Murital hamlet in Inaworena village on Tepala river, a 20% incidence of goitre was noted. Three cretinoid dwarfs were seen in this village. From inquiries made, swellings in the neck have been common for generations. In only one other village among 26,000 natives has goitre been found in significant numbers. It would seem advisable to attempt removal of the village and administration of iodized salt.

Epidemic Disease.

During 1947 Gollala, as did almost the whole territory, suffered considerable mortality from an influenza epidemic with subsequent secondary bronchopneumonia. The effect is reflected in the relatively few children in the one to five years age group in the population. There are multiple small foci of bacillary dysentery with very low mortality rate scattered throughout the district. Bacillary dysentery in natives responds very quickly to sulphaguanidine with much smaller dosage than is given to Europeans. *Granuloma venereum* reaches minor epidemic proportions after dances, to which people gather from villages up to two to three days' walk away. A dance lasting two months provides ideal conditions for dissemination of granuloma. Gonorrhoea is not prevalent, but cases with complications such as orchitis, epididymitis and peritonitis occur.

Skin disease is very common. Yaws mainly in the primary stage increases in incidence during the wet season. The incidence of primary yaws falls most heavily on young children and young adults. Gangosa was confined to adults. One case of tertiary bony yaws with pathological fracture and one with secondary papular eruption were encountered.

The high incidence of tinea imbricata and tinea circinata is shown in the analysis of diseases below. *Tinea imbricata* appears as if it might be related more to a nutritional deficiency than to infectivity of a fungus. Many cases were encountered in which close contact within a family group was ascertained, yet one member was suffering from universal tinea imbricata and the contact was quite free. Babies and children of infected mothers exhibited a clean skin. Scabies and secondary infection of lesions were relatively common. Superficial examination of the oral cavity, the mucous membrane and dentition gave the impression that dental caries is far less common than is reported in European communities. The periodontal tissues were unhealthy. The latter contrast is said to be related to an inverse ratio between acidogenic and proteolytic enzyme systems. Low caries incidence is thus associated with a high level of periodontal disease.

The physical build of natives differs greatly from that of Europeans. Male adults seldom attain five feet six inches in height, and the average is at least three inches below that. The average weight of adult males is, at maximum, nine stone. Both males and females are solidly built, muscular development is good and obesity is completely absent. The rigorous nature of the country fosters the development of powerful thighs even in young children. Although the figures are somewhat invalidated by the

inability to estimate age accurately, a series of 244 consecutive blood pressure readings was made by mercury manometer and revealed that in no age group does the average systolic blood pressure exceed 121 millimetres of mercury. Little elevation in blood pressure is observed with age.

Population Trends.

Census taking has been carried out only since 1951. According to the general impressions of missionaries who have been living for at least thirty years continuously in the district, the population is decreasing in some villages, slowly increasing in others. Death is due mainly to disease introduced from coastal areas. Malaria contracted by labourers on the coast is often the cause of death on their return to high altitudes, where bronchopneumonia is common in debilitated natives. Influenza and dysentery exact a steady toll. However, Professor Keesing, Professor of Anthropology at Stanford University, California, is quoted in the nutrition survey of 1947 as considering optimism for the future justified when more than 40% of the population is under the age of fifteen years, and the report goes on to refer to a Papuan "low of 26% Delta division" for the year 1935. The similar figure calculated for Gollala was 35%. From births and deaths recorded in the last census there has been a decrease in population in the area of patrol.

Infant Mortality and Child Welfare.

Questioning the village people through police interpreters as yet forms an unsatisfactory means of unearthing the degree of maternal mortality, miscarriage and infant mortality. In one village where a reliable source of information—to wit, an educated mission boy—was questioned, it was suggested that abortion is common among the younger women of the villages, who are not willing to bear children until they reach the age of thirty years. Rumours of methods of procuring abortions or of the presence of abortifacient plants have never been confirmed. Infants whose mothers die early in their infancy are put to the breast of other nursing mothers in the village. If none is available, the child is fed on juice squeezed from crushed sugar-cane, finely pre-masticated sweet potato, and water in which potatoes and pumpkin leaves are boiled. Children receive, at an early age, bulky dietary supplements such as baked sweet potatoes. Infants and young children are well nourished and healthy. The ten to fifteen years age group seems to be in a rather less fortunate nutritional state. It is gratifying to note that many pregnant women living close to Tapini have recently begun to come voluntarily to hospital for delivery. This points to some change in the attitude towards medicine.

The exact attitude of natives towards medical benefits is not easy to define. Treatment is given without charge, patients and friends are given daily rations at the hospital. Ample supplies of antibiotics are made available to the hospital. To the observer of but ten weeks it seems that uneducated natives are quite unable to comprehend on what basis they are being given free treatment. It may almost be said that they believe they are performing an admirable service to the Government by allowing themselves to be treated. Gratitude shown towards medical officers is rare. In the early days of the settlement a few errors of judgement were made by officers, but these have been rectified of late. In such a vast district to be controlled by one man, it is impossible to bring frequent contact to these people. Until staff shortages are relieved, improvement in the numbers of patients treated will be slow. Progress is being made and the future holds much promise.

To consolidate the advances made by the medical officer it is hoped that within a few years a broad education programme will be undertaken. Little can be achieved in the field of sanitation and personal hygiene until by example and instruction the minds of these people have been rendered capable of response by elementary education. At present there are four distinct language groups, and no form of writing is known. Agricultural improvements, the importing of livestock to supplement meagre protein supplies, health regulations controlling recruitment of highland natives for coastal labour gangs and a programme of education—all must be developed side by side with medical patrols and medical aid posts. Therein is the basis for future development of this evolutionarily backward people to a higher grade in the scale of civilization.

Inspection of Villages.

In order to facilitate the difficult task of collecting all natives at a central meeting place for examination, a native policeman is customarily sent ahead of the patrol to instruct

scattered hamlets to forgoth close to the route of the patrol. This often entails journeys of two or three hours for man, woman and child. It is not surprising, then, that some comply with directions only with reluctance. Invariably a small percentage of the population is absent visiting or in employment on coastal plantations. Thus it was possible to inspect no more than 74.3% of a population of 1398.

Natives were lined up in villages, females in one column, males in another. One may readily gain a general impression of the nutritional state and separate the obviously ill. After the recording of census statistics, each individual is examined for frambesia, tropical ulcer, skin diseases, eye disease, and vitamin and mineral deficiency. Children are palpated abdominally to detect splenomegaly. The occurrence of deaths and births must be elicited by an arduous interrogation per medium of police interpreter, the village constable and the family representative. Rechecking reveals discrepancies in the opinion given by different members of the village. Patients who never before have seen a medical patrol submit readily to treatment.

On the patrol reported here, a total of 13 villages were inspected between January 12 and January 28, 1952. This inspection entailed walking some 170 miles over country of which the topographical features were described earlier. Out of a total population of 1398, 1039 people were examined. An analysis according to age groups is given in Table II.

TABLE II.
Analysis of the Total Population in Age Groups.¹

Age Group.	Number.			Deaths.		
	Males.	Fe-males.	Total.	Males.	Fe-males.	Total.
Up to 1 year ..	23	14	37	9	4	13
1 to 5 years ..	45	62	107	5	4	9
5 to 10 years ..	89	67	156	—	5	5
10 to 15 years ..	106	88	194	2	—	2
Adults ..	484	420	904	28	21	49
Total ..	747	651	1398	44	40	84 ²

¹ There were 20 pregnant women.

² Total number of deaths since the last census.

The following is an analysis of the diseases present:

In the total population of 1398 persons, the incidence of the various diseases was as follows:

Frambesia: There were 32 adults (20 male and 12 female) and 14 children (12 male and two female) affected—a total of 46 persons (3.3%).

Tropical ulcer: No examples of tropical ulcer were found.

Skin disease: There were 32 people affected with scabies, 275 with tinea and one with another form of skin disease—a total of 298 persons (22%).

Tuberculosis: No tuberculosis was found.

Leprosy: No leprosy was found.

Eye disease: There were three cases of active and six of non-active eye disease—a total of nine (0.64%).

Hookworm disease: No hookworm disease was found.

New Guinea mouth: No New Guinea mouth was found.

Nutritional and deficiency diseases: No beriberi was found. There were 12 cases of goitre (five male and seven female subjects)—an incidence of 0.85%.

Among children only, no active malaria was found, but the spleen was palpable in 86 (61 males and 25 females)—an incidence of 17.2%.

W. S. EGERTON,
Brisbane.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Royal North Shore Hospital of Sydney, Crow's Nest, New South Wales, on April 22, 1954. The meeting took the form of a series of clinical demonstrations by members of the medical and

surgical staffs of the hospital. Part of this report appeared in the issue of July 31, 1954.

Gross Intracardiac Calcifications.

Dr. G. E. BAUER showed a married woman, aged seventy-nine years, in whom unusual cardiac signs had been discovered during routine medical examination several weeks before. Specific interrogation revealed no symptoms of heart disease; in particular the patient denied any chest pain or undue dyspnoea. There was no history of rheumatic fever or chorea in childhood. Examination of the patient showed the following signs in the cardio-vascular system. The pulse had a rate of 80 per minute with ventricular premature contractions; the vessel wall was palpable. The blood pressure was 135 millimetres of mercury, systolic, and 85 millimetres, diastolic. There was no evidence of congestive failure, the lung bases were clear, the liver was just palpable, and no peripheral oedema was present. The apex beat was palpable in the fifth left intercostal space, three and a half inches from the mid-line. A faint systolic thrill was palpable at the apex. The first heart sound was replaced at the apex by a loud Grade IV systolic murmur conducted over the entire precordium and to the axilla, with a short mid-diastolic murmur at the apex, harsh in character. No separate aortic murmurs were heard. Examination of the ocular fundus showed arteriosclerotic changes. The electrocardiogram showed a right bundle branch block pattern with pathological Q waves in leads I, aVL and V3, a prolonged P-R interval and ventricular premature contractions. Fluoroscopy showed an enlarged heart shadow, which was square-shaped, and massive calcification in the region of the atrio-ventricular ring, which moved synchronously with the heart beat. The appearance in the right anterior oblique view suggested a sheaving effect along the anterior heart border with abnormal pulsation probably due to ventricular aneurysm. (The screening was carried out in conjunction with Dr. Hatfield.)

Dr. Bauer said that the patient had massive intracardiac calcifications, especially in the region of the atrio-ventricular ring. There was no generalized disorder of calcium metabolism (normal serum calcium and blood inorganic phosphorus and phosphatase values were found) and the condition seemed to be due to local degenerative changes. The abnormal physical signs were apparently due to deposition of calcium on the mitral valve. Intracardiac rupture of chordae tendineae might also have occurred. Calcification had involved the conducting mechanism of the heart. Electrocardiographic and fluoroscopic changes suggested that the patient had had a silent cardiac infarction resulting in a ventricular aneurysm.

Wolff-Parkinson-White Syndrome.

Dr. Bauer also showed a woman, aged thirty-seven years, who had been suffering for several years from severe hypertensive heart disease, punctuated by recurrent episodes of pulmonary oedema. Left bundle branch block had been discovered over four years before. When first examined on October 14, 1952, the patient had shown evidence of left ventricular enlargement, gallop rhythm and a bigeminal heart action. The blood pressure was 240 millimetres of mercury, systolic, and 150 millimetres, diastolic. The patient was taking no drugs at the time, and there was no history suggestive of paroxysmal tachycardia, flutter or fibrillation. An electrocardiogram taken that day showed a unique tracing, namely, Wolff-Parkinson-White complexes alternating with left bundle branch block complexes. An electrocardiogram taken three days later showed the usual left bundle branch block. The patient was subsequently stabilized with parenteral hexamethonium bromide therapy and had kept very well and fully active during the past eighteen months without ever showing a recurrence of the Wolff-Parkinson-White complexes.

Dr. Bauer said that the Wolff-Parkinson-White syndrome (a short P-R interval with a prolonged QRS complex) was a cardiac conduction defect often seen in otherwise healthy individuals who were subject to attacks of paroxysmal tachycardia. More recently the association of Wolff-Parkinson-White syndrome with more serious types of heart disease had been stressed. Among the innumerable theories put forward to explain the condition the two most widely accepted were: (i) anomalous atrio-ventricular conduction due to an accessory conducting bundle (bundle of Kent)—in other words, a congenital anomaly; (ii) accelerated conduction across the atrio-ventricular nodal tissue probably due to ischaemic changes. Intermittent Wolff-Parkinson-White complexes had been described in the literature, but in all those instances the alternate beat was a normal sinus

complex. The present instance was a unique tracing of Wolff-Parkinson-White alternating with left bundle branch block, occurring in a patient with well-established heart disease.

Causalgia following Bilateral Wrist Fractures.

Dr. R. G. ROBINSON showed a woman, aged sixty years, who on October 31, 1953, had fallen, sustaining fractures of the right radial styloid and the left scaphoid bone. Because of impaction and the situation and type of the fractures, they were not observed in the X-ray films at the time. The pain persisted along with known pre-existing stiffness of the fingers. On November 17 X-ray examination revealed the fractures. Plaster of Paris splints were ordered to be applied bilaterally from well up on the forearm to the metacarpal heads. During the month following she complained of burning pain, worse at night and somewhat relieved by warmth, along the ulnar border of the right forearm. The plaster appeared satisfactory. On December 15 when the splints were removed, she suffered an immediate exacerbation of pain in both arms. This was evidently causalgia in nature. The splints were reapplied. On December 22 the fingers were dry, warm, edematous and stiff, movement being very painful. The hands and wrists were warm, tender and swollen. There was slight cyanosis with some atrophy. A diagnosis of Sudeck's atrophy was made. Neurosurgical opinion was against sympathectomy. By February 16, 1954, well-established second stage Sudeck's atrophy was present, with cyanosis, atrophy, early fibrous tissue contraction and osteoporosis of the bones of the wrists, hands and fingers. The pain was commencing to move up the arm, and there was slight stiffness with pain on movement of the elbows and shoulders. She was treated immediately, in addition to her splinting and physiotherapy, with oral cortisone therapy for three weeks. Initially 100 milligrammes were given, and after three days 50 milligrammes daily. There was great relief followed one week after cessation by an exacerbation. Twenty-five milligrammes of cortisone given by mouth twice daily had caused a second remission of symptoms and signs. It was anticipated that she would make a reasonable recovery on that regime.

Dr. Robinson said that de Takats postulated three neuron levels in the mechanism of this complication: (i) sensory neuron with persistent axon reflex; (ii) spinal neuron; (iii) cerebral neuron. Trauma activated vasodilator and vasoconstrictor mechanisms, leading to increased capillary pressure with exudation into the tissues, and also stimulation of sensory receptors. Luvis had shown the liberation of a painful substance at the termination of nerve fibres from the posterior root ganglia (nocifer nerves). Unless this stimulation was blocked or the substance neutralized the circuit would be completed with consequent sensitization of higher neurons and a spread of the condition. Since the internuncial pool was a closed series of synapses, once it was activated, it might continue to send a series of impulses down the nocifer nerves, thus maintaining the status. If that was so, then sympathectomy was not likely to result in any startling improvement. Cortisone was buffering the effects probably by decreasing capillary permeability and the progressive fibrosis. It was known that it did not affect the histamine response. The increased circulation might be leading to removal of the painful substances, thus breaking the cycle. Rheumatic conditions had been excluded by examination of the blood erythrocyte sedimentation rate and serum protein values.

Cardiac Investigation Clinic.

A series of patients were presented from the Cardiac Investigation Clinic (Dr. F. A. E. LAWES, Dr. R. D. PUFFETT, Dr. D. S. STUCKEY, Dr. R. G. EPPS).

Mitral Stenosis: Systemic Hypertension.

The first patient, a married woman, aged thirty-five years, had been well until 1944, when she had an attack of rheumatic fever (painful swollen joints, oedema of the legs and face, and breathlessness) while six weeks pregnant; she recovered in three months and had a normal delivery. Since then she had had intermittent swellings of the legs and exertional dyspnoea. During her fifth pregnancy in 1948 she became very breathless and had swelling of the legs, but carried the pregnancy to full term with normal delivery. Following this her symptoms lessened a little. In May, 1953, she again became pregnant; and, as her breathlessness greatly increased, pregnancy was terminated, and the

patient was sterilized in July, 1953. In December, 1953, she was moderately breathless on exertion and was able to do light shopping and housework. She had a sharp pain in the centre of her chest on exertion, relieved by rest, and also suffered from a continuous aching pain in both arms. Examination at that time showed slight central cyanosis. The pulse was regular with a well sustained pulse wave. The jugular venous pressure was two centimetres; the blood pressure was 150 millimetres of mercury, systolic, and 115 millimetres, diastolic; the right ventricular pulsation was increased. The first sound was increased in intensity at the apex, and the second sound was loud at the pulmonary area. A soft opening snap was heard with a soft systolic click at the pulmonary area. There was a mitral mid-diastolic murmur with presystolic accentuation. No systolic murmur was present nor were there any aortic murmurs. There was no oedema of the legs, enlargement of the liver or pulmonary congestion. Screening examination of the heart showed a greatly enlarged pulmonary artery shadow and small aorta. The lung fields appeared moderately congested. The right ventricle was enlarged, as was the left atrium. Calcification of the mitral valve was seen. An electrocardiogram showed marked pulmonary P waves, with right ventricular preponderance, after exertion to the point of dyspnoea, but not of pain; there was depression of ST and T waves in lead II. When examined again in February, 1954, the patient said that she had had a very severe attack of precordial pain precipitated by exertion. Examination on that occasion showed the blood pressure to be 170 millimetres of mercury, systolic, and 120 millimetres, diastolic, and the opening snap at the mitral area was rather louder than before, but otherwise her signs were unaltered. It was considered that she was suitable to undergo mitral valvulotomy if one could be sure that she had not suffered from recent rheumatic activity.

Atrial Septal Defect.

The second patient from the clinic, a woman, aged forty-six years, had known since the age of seven years that a murmur was present in her heart. For thirteen years she had had moderate breathlessness on exertion. She had attacks of severe palpitation in which the heart beat rapidly and regularly, and in which her vision became dim. On examination the hands were cold, and the jugular venous pressure was raised six centimetres. The radial pulse was irregular with coupled beats. The pulse wave was of small amplitude and was sustained. The blood pressure was 150 millimetres of mercury, systolic, and 100 millimetres, diastolic. The apex beat was in the anterior axillary line. There was a forceful cardiac impulse with a right ventricular lift. There was no pulmonary artery pulsation. The second sound at the pulmonary area was closely split and slightly accentuated. There was a soft blowing systolic murmur at the aortic pulmonary and mitral areas, loudest at the pulmonary area. There was a short diastolic murmur at the mitral area. The liver was enlarged one finger's breadth but was not tender. There was no oedema. X-ray screening examination showed a large heart. The pulmonary arteries were very prominent and were pulsating. The right ventricle was moderately enlarged. The left ventricle was slightly enlarged; the left atrium was not enlarged. No valvular calcification could be seen. The electrocardiogram showed low voltage, right bundle branch block and depression of S-T and T waves in all leads.

Tetralogy of Fallot.

The last patient from the clinic was a boy, aged five years, in whom cyanosis had been noted at birth. He was very short of breath on exercise and could run only thirty yards; he had been observed to squat when exhausted. Examination of the boy showed marked central cyanosis and well-developed clubbing of the fingers and toes. The radial pulse was normal, as was the jugular venous pressure. There was increased right ventricular pulsation, with a grade 3 systolic murmur, maximal in the third left intercostal space at the sternal border, and loud second sounds at the pulmonary area. The electrocardiogram showed pulmonary P waves, right axis deviation and right ventricular preponderance. Fluoroscopic screening examination showed a slight increase in the general size of the heart, a small pulmonary artery shadow and lung fields which were clearer than normal. Operative treatment had been recommended.

Thoracic Unit.

A series of patients were presented from the Thoracic Unit.

Pulmonary Cyst in Pregnancy.

DR. BRUCE WHITE showed a married woman, aged thirty-eight years, who had been admitted to the Royal North Shore Hospital of Sydney on March 13, 1954, complaining of daily asthmatic attacks since December, 1953. She was seven months pregnant. Treatment was given with adrenaline in oil and aminophylline. It was learnt that she had had asthma since the age of seventeen years and that it had been severe during the previous pregnancy. X-ray examination in December, 1948, had shown a small air cyst in the lower lobe of the left lung. While the patient was in hospital the asthma subsided, but a persistent productive cough remained. Signs were present of an air cyst at the left lung base; these were confirmed by radiography. Because of the danger of rupture of the cyst in labour, with development of tension pneumothorax, bronchoscopy and then lobectomy were decided upon and carried out with no complications; the patient did not come into labour. She was transferred to the obstetric unit of the hospital on the seventh post-operative day, with severe pitting oedema of the ankle but no raised blood pressure. That was the patient's current condition.

Foreign Body in Bronchus Simulating Carcinoma.

DR. C. G. BAYLISS showed a male patient, who was aged fifty-seven years when first examined on August 10, 1953. He had had a cough with some sputum for several years, but this had not caused him much inconvenience until an attack of influenza about three months previously. Since then the cough had been worse, and there was more sputum, which was now offensive in character. He had noticed a wheeze in the upper part of the chest on the right side, and that was becoming more noticeable. Nothing of importance was elicited in his history at this stage. The main findings on examination were an audible wheeze over the right side of the chest and râles and rhonchi on auscultation over the base of the right lung. At this stage the findings were considered very suggestive of carcinoma blocking the right lower lobe bronchus. On August 11, 1953, bronchoscopy was performed by Dr. Ian Monk. Two nodules were seen just below the opening of the middle lobe bronchus. Carcinoma was thought likely, but examination of a biopsy specimen did not confirm this. On September 30 bronchoscopy was carried out and showed atelectasis of the lower and middle lobes of the right lung with bronchiectasis in the collapsed lobes. On October 20 a further bronchoscopy was performed by Dr. Monk. A large mass was now seen projecting from the right middle lobe bronchus. A biopsy specimen again did not show carcinoma.

Dr. Bayliss said that, whatever the cause, it was now felt that thoracotomy with resection according to the findings should be carried out. On November 4 the patient was admitted to hospital. His history was taken by the house physician on the day of admission and was in accordance with what was already known. However, on the following day he volunteered a piece of information which seemed very important. About November, 1951, whilst he was swallowing soup a sharp piece of bone about three-quarters of an inch in length had become stuck in his throat, and he had felt it at a point just below the clavicle about one inch to the right of the sternum. He had developed a cough which lasted some weeks. On November 25, 1953, thoracotomy was performed. The middle and lower lobes of the right lung were atelectatic and were removed. A piece of chop bone was found obstructing the right stem bronchus. Convalescence was uneventful, and the patient was now very well.

Cavernous Pulmonary Telangiectasis.

DR. A. G. McMANIS showed a woman, aged forty-seven years. She had had an X-ray examination of her chest in August, 1950, because of post-herpetic neuralgia, and that had revealed a lesion in the right lower zone. When she attended the Royal North Shore Hospital of Sydney on March 16, 1953, the X-ray appearances were unchanged. On examination of the patient it was noticed that she was slightly cyanosed and had telangiectases on the face, lips, tongue and roof of the mouth. She gave a history of having suffered from nose bleeding. The fingers were slightly clubbed. A loud continuous murmur was heard in the right axilla and to the right of the sternum; it was best heard on full inspiration. Both antero-posterior and lateral tomograms were prepared; these showed that there were two separate lesions on the right side, one in the anterior segment of the upper lobe and the other in the anterior basal segment; prolongation of this lesion to and from the mediastinum could be heard.

It was learnt that the patient's father had had similar telangiectases on his face, and suffered from nose bleeding. During his final illness he had been in the Royal North Shore Hospital of Sydney, and at that time his X-ray film showed an opacity which was reported as being a secondary neoplasm. At autopsy, he was reported as having a blood cyst of the lung. It was most probable that this was a similar condition to his daughter's.

It was decided to resect these vascular lesions, and on November 18 Dr. Monk removed them both, without removing any pulmonary tissue. She made a complete recovery from her operation, and her general appearance had improved.

Developmental Cyst of Lung.

DR. M. P. SUSMAN showed a married woman, aged fifty-one years, who, ten years prior to admission to hospital, had had a right *empyema thoracis* treated by drainage. Since then frequent "colds", shortness of breath and yearly episodes of "pneumonia and pleurisy" had occurred. Eight weeks prior to admission to hospital she developed cough with copious offensive greenish sputum. There was also recent weight loss and chest pain on the right side. X-ray examination revealed a cavity containing gas and fluid in the right paravertebral gutter at the level of the tenth and eleventh thoracic vertebrae, consistent with chronic empyema or lung abscess. At operation, a large intrapulmonary cyst of the lower lobe was found, purulent fluid aspirated, and segmental resection of the lobe performed, the apical segment only being left. One month after discharge from hospital the patient suffered from right-sided chest pain, fever and rigors. Blood-stained fluid was aspirated from the right pleural cavity. Thoracotomy was performed, a chronic hemothorax evacuated, and tube drainage instituted. The cavity was reduced to a ten-millilitre volume at the time of her discharge from hospital. Histopathological examination of the tissue showed in a section from the wall of the larger cavity a lining of granulation tissue with a few epithelial cells, muscle bundles running longitudinally and circumferentially, and the remains of mucous glands. The findings suggested a bronchial origin. The amount of muscle seemed excessive for the cyst to be mere ectasia of a normal bronchus. It was more probably of developmental origin. The smaller cyst presented the same appearances.

Units of Neurology and Neurosurgery.

A series of patients were presented from the Units of Neurology and Neurosurgery (DR. I. A. BRODZIAK, DR. G. SELBY, DR. ERIC DAVIS, DR. J. GRANT, DR. G. VANDERFIELD).

Erb's Juvenile Muscular Dystrophy.

The first patient, a married woman, aged forty-five years, had in 1933 noticed weakness of her back and thighs, causing her to fall if she walked quickly; when she walked she would tend to deviate towards one side. In 1949 she had noticed a similar weakness of her elbows. These symptoms had gradually progressed, causing her to become bedridden about May, 1953. She had noticed no symptoms referable to her cranial nerves, no involuntary movements, no sensory changes except for occasional occurrence of paresthesia in her feet, and no sphincteric disturbances or defects of coordination.

On examination, the patient was found to be unable to stand, and she had pronounced weakness of the proximal muscle groups of both limbs, with wasting of the gluteal and thigh muscles, and of the muscles around the shoulder joints. Fibrillation was not detected, and motor power in the muscles of the hands and feet appeared essentially normal. The patient denied any change of appearance in her face, but the facial musculature was weak and the smile of the transverse character usually seen with the Landouzy-Déjerine type of muscular dystrophy. Sensation was intact; the left knee jerk was absent, the right knee jerk was greatly diminished, and both triceps and biceps jerks were diminished. The ankle jerks were normal and the plantar responses flexor. Both sterno-mastoid muscles were normal, and no abnormality was found in any other system. The electrical reactions of the muscles were varied and inconstant, a finding often present in this condition. The comment was made that the early onset of symptoms and the course of the patient's illness were typical of the Erb's juvenile type of muscular dystrophy, though there was no clear-cut defining line between this and the Landouzy-Déjerine type. The patient was given exercises to all muscles, but little improvement was achieved.

Compression Neuritis of the Right External Popliteal Nerve.

The second patient was a married woman, aged thirty-seven years. In December, 1953, her husband had slept with his legs across her right leg; she awakened next morning with a feeling of tightness in the right foot, and soon afterwards became aware of a weakness of the right ankle and right foot drop. She complained also of paresthesia in the right great toe, dorsum of the right foot, and lateral aspect of the lower third of the right leg. The paresthesia had decreased, but the weakness had persisted. In March, 1953, after leaning with her right elbow on a railway window for some considerable time, she developed a right ulnar palsy, from which she recovered after a few months.

On examination, the patient was found to be a very thin woman with little covering over the bony prominences. She had right foot drop and paralysis of the evertors of the right foot and of the extensors of the right ankle and toes (the peronei, *tibialis anterior*, *extensor hallucis* and *extensor digitorum*). Moderate residual wasting of the right interosseal and hypothenar eminence was detected with corresponding weakness of the muscles and of the right *flexor carpi ulnaris*. Superficial sensation was impaired over the lateral aspect of the lower third of the right leg, and the dorsum of the right foot (the distribution of the external popliteal nerve). The knee jerks were brisk and equal; the right ankle jerk was absent, the left present. The findings from a full blood count were normal. The result of the Eagle flocculation test was negative. The fasting blood sugar level was 114 milligrammes per 100 millilitres. X-ray examination of the knee joints revealed bilateral *osteocondritis dissecans* with large loose bodies in the joint spaces and moderate osteoarthritic changes. X-ray examination of the lumbosacral part of the spine revealed *spina bifida occulta* in the first sacral vertebra. The response to faradic current applied to the affected muscles was weak, the test confirming the presence of a lesion of the external popliteal nerve. The diagnosis was a compression lesion of the right external popliteal nerve.

Spontaneous Intracerebral Hematoma.

A woman, aged forty years, had been treated recently for an ear infection. On February 27, 1954, she became stuporose and was admitted to the Royal North Shore Hospital of Sydney, where she was found to have a temperature of 101° F., a right visual field defect, slight dysphasia and increased reflexes on the right side of the body. The leucocyte count was 11,000 per cubic millimetre, and the cerebro-spinal fluid contained 17 cells per cubic millimetre and a protein content raised to 30 milligrammes per 100 millilitres. Those findings led to strong suspicion of cerebral abscess. Left carotid angiography showed a shift, suggesting a space-occupying lesion on the left side, but not assisting otherwise in localization. The making of exploratory burr holes in the left temporal and left parietal regions failed to reveal any abscess or other abnormality there, and it was noted that intracranial pressure was not increased and that there was a large left temporal horn. Later, air studies confirmed the presence of a shift and showed that it was due to a space-occupying lesion in the left occipital lobe beneath the floor of the occipital horn of the lateral ventricle. At craniotomy on March 19 a posterior osteoplastic flap was turned down, and through the occipital horn of the ventricle the lesion was approached. It proved to be an intracerebral hematoma of moderate size, and after evacuation no abnormality could be seen in the walls of the cavity to account for its presence. Presumably a vascular abnormality was responsible. Recovery from operation was satisfactory, although right homonymous hemianopia was still complete. Post-operative air studies showed restoration of the left occipital horn to a normal position.

Traumatic Arachnoid Cyst.

An adult male patient presented with a history of "queer turns" for a period of twelve months. In the turns his surroundings would appear to recede from him, and objects and people would appear to become distant. There was a feeling of unreality associated with the episodes. As well as the minor turns he had had three major attacks of convulsions. The significant feature in his previous health was that as a child he had suffered from a head injury which was associated with a linear fracture in the right parietal region. The only abnormality found on clinical examination of the patient's central nervous system was a suggestion of a left inferior homonymous field defect. Palpation of the skull revealed a bony defect in the right parietal region. X-ray examination of the skull confirmed the presence of this defect, which was associated with a linear fracture extending upwards to the vertex. Right carotid angiography

indicated a vascular displacement suggesting a space-occupying lesion underlying the bony defect. Pneumoencephalography showed a dilated ventricular system in relation to the bony defect, and subarachnoid air also in this region. Charting of the visual fields confirmed the visual defect. Full cerebro-spinal fluid examination showed no abnormality. Electroencephalography showed a prominent delta wave abnormality again in the region of the defect and also well-marked spike activity in this region. At operation under general anaesthesia, a meningo-vascular scar was excised which connected the scalp tissues to the cerebral cortex. Associated with this was found a subarachnoid collection of yellow fluid. The gyrus underlying the anterior part of the defect was coagulated, as this corresponded to the area from which the spike focus was probably arising. The dural defect was closed with *fascia lata*. The bony defect was closed with a tantalum plate. Post-operatively the patient's condition remained satisfactory. He took "Dilantin" and "Prominal" and had no further seizures. Post-operative electroencephalography showed that the spike focus had disappeared.

Extradural Hematoma.

An adult male patient was transferred to the Royal North Shore Hospital of Sydney on March 16 from Wallend Hospital with a history of having received a head injury on March 4. He had been unconscious on admission to Wallend Hospital, but on regaining consciousness showed evidence of motor aphasia and a right spastic hemiparesis. His pulse rate had been noted to be slow, and he had vomited on several occasions. Lumbar puncture was said to have shown the cerebro-spinal fluid to be under increased pressure. Examination on admission to the Royal North Shore Hospital of Sydney confirmed the previous clinical findings. Lumbar puncture showed a pressure of 190 millimetres of cerebro-spinal fluid with no increase of protein. X-ray examination of the skull showed a linear fracture involving the left squamous and temporal and parietal bones. Left carotid arteriography on March 17 revealed a displacement of the middle cerebral vessels away from the bones of the skull, and it was felt that this indicated the presence of a subdural hematoma. At operation under local anaesthesia on March 19 a large extradural hematoma was found. This almost completely covered the temporal and parietal regions. Post-operatively, he showed a steady improvement. The comment was made that on review of his arteriogram it was felt that possibly the diagnosis of an extradural hematoma could have been made, as there was a suggestion in the antero-posterior films of filling of the middle meningeal artery which was also displaced medially along with the middle cerebral vessels.

Cervical Disk Lesion Associated with Muscular Dystrophy.

The last patient, a man, aged forty-two years, when first interviewed at the neurology out-patient clinic, had complained of (i) stiffness and weakness of his neck and stiffness and weakness of his shoulders since adolescence, (ii) progressive weakness of his left arm and left leg for nineteen years, (iii) numbness of his right leg for nineteen years, and (iv) burning pain in the back of his neck radiating down into both arms for nineteen years. The family history was non-contributory. The patient had first noticed evidence of his muscle dystrophy at the age of seventeen years. The onset was gradual with slowly progressive weakness and wasting of his facial musculature, followed by weakness of the neck and of muscles of the shoulder girdles and arms. Reduction in mobility of the shoulder joints gradually followed the dystrophy. In 1935 he was involved in a car accident, when he received a blow on the back of his head, neck and shoulders. That appeared to accelerate the weakness and stiffness of his shoulders, and subsequently he developed a slowly progressive weakness of his left leg (distal more than proximal) and paresthesia and numbness of his right leg. During this period he had severe burning pain in his neck radiating down both arms. He was admitted to the Royal North Shore Hospital of Sydney for investigation. The diagnosis on admission was muscle dystrophy and cord compression with partial Brown-Séquard syndrome. The following salient features were recorded on physical examination of the patient: (i) facio-scapulo-humeral dystrophy (Landouzy-Déjerine type); (ii) torticollis with the head flexed to the right and chin pointing to the left; (iii) evidence of cord compression at the fifth cervical spinal segment, atrophic paralysis of rhomboids, deltoids, infraspinati and supraspinati, biceps and *supinator longus* muscles, more on the left than the right, spastic weakness of the remaining musculature of the upper limbs, more on the left than the

right, exaggeration of biceps jerks and triceps jerks on both sides, and inversion of radial reflex on both sides; (iv) evidence of upper motor neuron lesions—namely, spastic weakness of the left lower extremity with exaggerated deep reflexes and an equivocal Babinski response on the left side; (v) crossed hemianesthesia and hypoaesthesia on the right side of the body up to approximately the seventh cervical spinal segment, and ipsilateral blunting of tactile sensation and muscle sensibility together with impaired vibration sense over the left lower extremity; (vi) posterior column ataxia of the lower extremities.

Plain X-ray examination of the cervical part of the spine showed that the disk between the sixth and seventh cervical vertebrae was irregular in outline; there was much reactive bone formation anteriorly and posteriorly in relation to this disk, with rotational deformity and loss of curvature at the affected joint. Myelography was carried out by introducing six millilitres of "Pantopaque" by the lumbar route and making a screening examination of the patient on the tilting table. The dye flowed freely through the lumbar and dorsal canals into the cervical canal. At the level of the fifth and sixth cervical vertebrae extending to the upper border of the seventh cervical vertebrae there was an obstruction to the flow of the dye in the mid-line, the dye flowing upwards along both lateral gutters leaving an approximately oval mid-line filling defect maximal over the disk space between the fifth and sixth cervical vertebrae. In the lateral view a complete interruption of the column was seen opposite the posterior border of the disk space between the fifth and sixth cervical vertebrae, but no abnormality at the other cervical disk levels. It was concluded that an advanced mid-line disk space protrusion was present between the fifth and sixth cervical vertebrae. Examination of the cerebro-spinal fluid revealed the following protein content: 40 milligrammes per 100 millilitres; sugar content, 60 milligrammes per 100 millilitres; globulin content, no increase; cells, nil; Wassermann reaction, negative; colloidal gold test, no reaction.

The patient was examined in consultation by Dr. J. M. F. Grant, who agreed that the patient had a chronic disk protrusion causing cord compression. It was thought that the primary muscle dystrophy had allowed distortion and angulation of the cervical part of the spine and secondary spondylosis with disk degeneration and protrusion. Laminectomy and spinal fusion were recommended.

Cerebral Angioma.

Dr. J. M. F. GRANT showed a married woman, aged nineteen years, who had been admitted to hospital on May 2, 1953, with a diagnosis of subarachnoid haemorrhage. She had been unconscious prior to admission, and on examination was found to be drowsy with a complete left hemiplegia and hemianesthesia. Lumbar puncture showed blood-stained cerebro-spinal fluid under high pressure. Arteriography was carried out, which demonstrated the presence of an angioma in the right parietal region. The appearance also suggested the presence of a haematoma situated anteriorly to the lesion. Investigation of her previous history revealed that she had suffered from two previous subarachnoid haemorrhages, and that for many years she had suffered from epileptic seizures. On June 13 craniotomy was carried out; the haematoma was evacuated and the lesion excised. Her convalescence was stormy; on June 16 an extradural haematoma was evacuated, and on July 20, owing to a wound infection, the bone flap was removed. After this procedure she made a steady improvement and was at the present time able to carry out her normal household duties. She was discharged from hospital receiving medication of "Dilantin" and "Prominal" and had had no further seizures.

Arterio-Venous Fistula.

Dr. Grant also showed a married woman, aged forty-four years, who gave a history of severe headaches, deafness and a pulsating noise in the right ear for nine months. Recently she had become breathless and had been treated for congestive cardiac failure. The only possible significant features of her previous health were a history of a head injury some twenty years previously when she was struck on the right side of the head by a clothes prop. Following this injury she was treated for what was described as a nervous condition, in which she was confused and appeared to have lost the use of her limbs. However, she made a good recovery from this and evidently had had no other symptoms until nine months before the present time. Examination of the patient revealed a large vascular channel in the right occipital region, over which there was a palpable thrill, and a loud bruit could also be heard in this region. The bruit was diminished by compression on the carotid artery in the neck. This was associated with a feeling of faintness and

a considerable falling of the pulse rate. A blood pressure reading of 180 millimetres of mercury, systolic, and 80 millimetres, diastolic, was obtained, and she had mild cardiac enlargement. It was considered that the patient probably had an arterio-venous fistula between either the occipital or posterior auricular artery and, possibly, the lateral sinus. It was intended to investigate the condition by means of arteriography to see whether any possible surgical treatment could be undertaken, as, unless that was feasible, the prognosis appeared to be poor.

Hemiplegia following Neck Trauma.

DR. G. VANDERFIELD (with Dr. T. F. ROSE) showed a man, aged thirty years, who had been admitted to the Royal North Shore Hospital of Sydney in July, 1953, within half an hour of injury in a car accident near by. Although he had evidently not received a severe head injury and was conscious and alert, he was aphasic and had complete right hemiplegia. There was a deep wound on the left side of the neck, and the left mandible was fractured; also there was a compound fracture of the left tibia. On the same night the left internal carotid artery was explored up to the base of the skull. Bone fragments were found about the carotid sheath, but there was no obvious damage to the artery, which was pulsating normally. At the same time the sympathetic trunk was blocked. The fractured tibia was reduced, and after closure of the wound the leg was immobilized in a long leg plaster. Operation was followed by frequent stellate blocks, and a course of papaverine was given; but there was no immediate improvement in relation to the hemiplegia, presumably because of thrombosis of the left side of the circle of Willis. Two weeks later left carotid angiography showed moderate filling of the left internal carotid and middle cerebral arteries and poor filling of the left anterior cerebral artery; thus the vessels had evidently been reopened. Subsequently, with the assistance of physiotherapy and speech therapy, there had been fair recovery of use of the right leg, minimal recovery in the arm, and some return of speech, but it now seemed that a considerable degree of the hemiplegia and aphasia would be permanent.

Gunshot Wound Penetrating Abdomen and Involving Cauda Equina.

Dr. Vanderfield (with Dr. L. S. LOWWENTHAL) next showed a boy, aged eleven years, who had been admitted to the Royal North Shore Hospital of Sydney on February 3, 1954. He had been accidentally shot with a 0.22 bullet forty-eight hours earlier in the country. The bullet had entered just to the left of the xiphisternum and emerged about half an inch to the left of the spine of the first lumbar vertebra. All spinal cord functions below the level were immediately lost, but his general condition was surprisingly good. He had vomited once or twice, but there were no other abnormal findings apart from local tenderness about the wounds of entry and exit. X-ray examination showed fracture of the left lamina and pedicle of the first lumbar vertebra. A lumbar puncture yielded freshly blood-stained fluid, which rose on jugular compression but did not fall at all, indicating partial spinal blockage. Immediate laminectomy was decided upon. The fragments of bone in the bullet track were removed together with extradural blood clot. The dura itself did not appear to be lacerated; but when it was opened the cerebro-spinal fluid was found to be blood-stained, and the lower end of the spinal cord was contused. The roots of the cauda equina appeared intact. Good decompression having been provided, the wound was closed. The boy made a good recovery from the operation, and his general condition remained good. Since operation slow but continuous recovery of neurological functions had been proceeding, and he now had good sensation down to both knees and in the bladder, which had become reflex in action. There was fair power proximally in the right leg, and some voluntary movements were beginning in the left.

Medulloblastoma of Cerebellum.

Dr. Vanderfield's last patient was a boy, aged twelve years, who had been admitted to the Royal North Shore Hospital of Sydney on August 28, 1952. He gave a three months' history of vomiting and headaches, particularly vomiting, and not much else. On examination he was found to have bilateral papilledema, some left cerebellar ataxia and nystagmus on looking to the right. Also his right arm was a little unsteady in some of the tests. Preliminary ventriculography showed symmetrical internal hydrocephalus of moderate degree. The third ventricle was filled, but nothing appeared in the fourth ventricle. This confirmed the diagnosis of cerebellar tumour, and operation proceeded under general anaesthesia. The bone of the posterior fossa was removed; and as a result of

needling through the dura, tumour tissue was obtained. Dr. Viner Smith reported it to be medulloblastoma tissue. After adequate decompression had been provided by wide removal of bone, including the arch of the atlas, the wound was closed. Satisfactory recovery from the operation took place. Deep X-ray therapy was given to the cerebellum and spine. He was discharged from hospital on December 21, 1952, by which time all symptoms had cleared and the papilloedema had subsided. He had returned to school within a few months and had remained well.

Congenital Oesophageal Atresia.

Dr. ERIC GOULSTON presented a baby who had been suffering from congenital oesophageal atresia. The baby had been born at the Royal North Shore Hospital of Sydney on October 24, 1953. The mother's pregnancy had been associated with bleeding at three months and hydramnios. The baby was noticed to have an excess of mucus and to suffer from vomiting and cyanosis on being fed. A catheter could not be passed into the stomach, and X-ray examination with "Lipiodol" showed that the oesophagus ended in a blind pouch. Air was noted in the bowel. After pre-operative preparation, operation was performed on October 25 with a right retropleural approach and removal of the fourth rib. The tracheo-oesophageal fistula was ligated and cut, and an end-to-end oesophageal anastomosis was made. The anaesthesia induced by Dr. J. F. McCulloch was with ether, gas and oxygen, and "Scoline". Recovery from operation was good with gradual resumption of oral feeding after four days. An X-ray examination with "Lipiodol" swallow on November 9 showed a stricture of the oesophagus one centimetre in length at the level of the third thoracic vertebra. A dilatation of this area was performed by Dr. J. Dove two months later when the mother noticed some feeding difficulty, and the child had been symptom-free since.

Crohn's Disease of the Rectum.

Dr. Goulston then showed a married woman patient who had been admitted to hospital in December, 1953, complaining of rectal haemorrhage for six weeks and loss in weight of half a stone. There was no change in bowel habits. The results of abdominal examination were negative, but rectally a large polypoid mass was palpable, and proctoscopically this was seen to be seven centimetres from the exterior and considered to be fixed deeply to a hard indurated area. From a previous barium enema examination it had been reported that there was a constant filling defect in the left lateral portion of the rectum two and a half inches above the orifice, very suggestive of neoplasm. No evidence of obstruction was found. No biopsy was performed, but a confident diagnosis of carcinoma of the rectum was made. Abdomino-perineal resection was performed in due course. Macroscopically the polypoid tumour was seen to be attached with a wide base to a very thickened and indurated rectal wall, and there were several lymph glands involved near by. Microscopically the polypus was found to be benign. The thickening of the rectal wall was due to a chronic inflammatory process in all coats. Foci of inflammatory cells with giant cells were present with considerable fibrosis. Several lymph glands contained prominent collections of histiocytes and giant cells. It was considered that the thickening was due to Crohn's disease. Subsequently the perineal wound was very slow to heal, but when cortisone therapy was commenced there was great improvement.

Hypertrophic Pyloric Stenosis in an Adult.

Dr. Goulston's last patient was a man, aged forty-three years, who had been admitted to hospital on December 10, 1953, with a diagnosis of pyloric stenosis. He gave a history of loss of appetite, intermittent vomiting and loss of four stone in weight in the past eighteen months. He had had haematemesis and melaena in 1951, and after this episode had suffered from abdominal discomfort and distension five to ten minutes after taking food. During 1953 he had had another haematemesis, which required a blood transfusion. His family and previous histories provided no significant information. On examination, the patient looked as if he had lost the stated amount of weight from twelve stone to eight stone. He had fullness in the epigastrium, and there were visible and palpable peristaltic waves passing right across the upper part of the abdomen. There was no tenderness in the abdomen, and no masses were palpable. His other systems were normal. The hemoglobin value was 14.2 grammes per centum. After barium X-ray examination the following report was made: "The stomach is dilated with retained fluid and secretions and there is a high-grade pyloric stenosis. Practically none of the meal had left the stomach in five hours. The appearance suggests stenosing

duodenal ulcer, but the exact nature of the pathology could not be outlined." The free hydrochloric acid was greatly reduced in the test meal findings. Operation was performed on December 23 after suitable pre-operative preparation, and a grossly thickened pylorus was found. A partial gastrectomy was performed, and the patient made an uninterrupted convalescence. The histopathological report showed that the constriction was localized to the pyloric canal, where the great thickening was due to widening of the muscular coat. The mucosa and submucosa appeared normal. There was no fibrosis, and this was taken to exclude pyloric stenosis due to chronic peptic ulceration. The condition was an example of the adult form of hypertrophic pyloric stenosis.

Dr. Goulston commented that the aetiology of hypertrophic pyloric stenosis in adults was not clearly defined. The presence of symptoms from infancy was strongly suggestive of the congenital origin. However, the picture was uncommon, and most cases in adult life were associated with peptic ulceration, or gastritis, which was secondary to the stenosis and stasis. The primary pathological change was hypertrophy and hyperplasia of the circular pyloric muscle, and superficial as well as deep ulcers and inflammatory changes were often found. There was no characteristic finding to differentiate the condition from other pyloric conditions, and surgical treatment was indicated.

(To be continued.)

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

A DEATH FROM CHOLERA.¹

[From *The Sydney Morning Herald*, February 15, 1851.]

ABOUT one o'clock on Thursday afternoon a butcher named William Reardon residing in Essex St. was taken unwell and the illness increasing at eleven o'clock at night Mr. Surgeon Nelson was called in who found him in a state of collapse suffering from marked symptoms of cholera. About 12 o'clock Dr. MacKellar was also called in and a few minutes after Reardon expired. Both professional gentlemen concurred in the opinion that the deceased died from cholera. A post-mortem examination confirmed this opinion—the medical men stating that they believed the disease to have been brought on by local causes. There was a large open drain full of putrid filth in the front of the house of deceased and a quantity of meat in an unwholesome state was on the premises. Dr. MacKellar and Mr. Nelson with praiseworthy promptitude immediately communicated the facts to Mr. McLedie, Inspector of Police, who caused the meat to be seized and destroyed and the premises cleansed with chloride of lime. The coroner made an enquiry into the circumstances of the case but did not think an inquest was necessary.

Special Correspondence.

PARIS LETTER.

By OUR SPECIAL CORRESPONDENT.

Crenotherapy in France.

THE object of this letter is to show the very large importance given to crenotherapy (treatment by water from mineral springs) under present conditions of French medical science. It is also intended to give briefly a general idea of this ancient and yet still modern medical weapon. Thermal treatment is used throughout Europe—in England, Italy, Spain, Germany, Austria, Union of Socialist Soviet Republics—but it has been said that France above all is the "country of thermalism".

¹ From the original in the Mitchell Library, Sydney.

Importance of "Thermalism".

The place of crenotherapy in France is shown by the following facts:

In each medical school there is a chair and laboratory of hydrology, and in Paris, in addition, an *Institut national d'hydrologie et de climatologie*. Crenotherapy is a compulsory subject of fifth year medicine with a special examination. Moreover, there are three independent scientific and technical bodies, the *Société d'hydrologie et de climatologie de Paris*, the *Société internationale d'hydrologie médicale*, and the big *Fédération thermale et climatique française*, which includes not only physicians but also representatives of the municipal councils, of spas and of the hotel industry. Every year numerous articles are published in specialized scientific journals, such as the *Annales de l'Institut d'hydrologie*, *Annales de la Société d'hydrologie*, and *Presse thermale et climatique*, or in special issues of ordinary medical journals. Every year medical theses are written on "thermalism", and a congress takes place in a thermal resort. Every three years the important *Congress international du thermalisme et du climatisme* publishes a lengthy report. Crenotherapy in France is a medical specialty with 600 physician specialists and 1200 physicians acting chiefly as "thermal doctors"—all grouped in an association, *Syndicat des médecins thermaux*. France has 1500 mineral springs in 100 thermal resorts, and more than 600,000 patients are treated every year. "Thermalism" in general is under the control of the *Commission permanente des stations hydrominérales et climatiques* of the Ministry of Health.

History.

Mineral waters have been used in Europe for medical treatment from earliest known times. They were regarded as being supernatural and religious phenomena, and pagan priests were the first hydrologists. In Greece Hippocrates rarely mentions mineral water, but the springs of the Greek Archipelago, Asia and Africa are praised by Pindar, Aristotle, Plutarch, Galen, Pausanias and others. The Romans were the first to canalize the waters, to build luxurious spas, and Herodote the Physician established the first medical principles of crenotherapy. In Gaul numerous springs are known to have been used long before the Roman conquest. Christianity at first disapproved, but little by little Christian saints were substituted for the former pagan divinities of the fountains, and many springs became the property of convents and religious orders. Thousands of wounded and sick Crusaders were received and treated in the spas of the Middle Ages. During the sixteenth century Ambroise Paré and Bernard Palissy tried to discover the origin of mineral waters, and the great philosopher Montaigne was a fanatical patient, travelling from one thermal resort to another throughout Europe.

During the seventeenth century thermal "cures" became extremely fashionable. The nobility of Louis XIV's court went "to take the waters", as related by Madame de Sévigné, and the first analysis of French mineral waters was made by the young Academy of Science. The Royal Society of Medicine collected in Paris during the eighteenth century innumerable scientific publications on thermal waters. The beginning of last century saw wounded soldiers of the Napoleonic Wars crowding to French spas. Later thermal towns became popular society resorts and were patronized by literary figures such as Chateaubriand and Lamartine, and many others. Moreover, King Louis Philippe's queen gave her name to Amélie-les-Bains, and the Empress Eugénie founded Eugénie-les-Bains. In the twentieth century great efforts were made to improve the amenities of the spas. Millions of soldiers of the two world wars were treated. Scientific and medical research has been powerfully developed and organization improved, and "social thermalism" has brought spa treatment within the reach of all.

Origin, Composition and Action of Mineral Water.

The quantity of thermal springs in France has a simple geological explanation—that is, diversified ground from every geological era, with numerous faults through which water from the depths comes to the surface. It is believed that mineral waters are of two different origins—namely, infiltration water and eruptive water. The first is due to rain seeping deep enough to become warm, dissolve minerals and rise to the surface through a fault. The temperature of this water is under 95° F.; its discharge varies according to weather and season.

Eruptive water on the contrary does not come from the atmosphere, but is formed by synthesis or by distillation of rocks in volcanic phenomena. The temperature is high, over 176° F. The water contains minerals which exist only

at great depth and is radioactive. Flow is independent of weather and season. Some sources are probably a mixture of the two types.

Physical Properties.—The temperature varies from 44·6° F. (Forges-les-Eaux) to 190° F. (Chaudesaigues) and even 203° F. (Hamman-Meskoutine, Algeria). The density reaches 1·16 at Salles de Bearn. The pH ranges from 6·7 to 9·7. Some waters are oxidizing, others are reducing, as at Vichy. Many gases are in solution—for example, carbonic acid, nitrogen, oxygen, hydrogen, sulphuretted hydrogen and also rare gases, such as helium, argon, krypton and xenon. Radioactivity is from 0·1 millimicrocurie per litre (Allevard) to 106 millimicrocuries (Chateldon).

Chemical Properties.—Mineralization varies from a few grains to nearly half a pound per litre. The results of first analyses were given in salt, but today we know that 90% of the elements are in the ionic form. The following elements are found in very variable concentration and association: chlorine, sodium, sulphur, carbon, oxygen, hydrogen, calcium, iron, magnesium, manganese, copper, arsenic, iodine, bromine, lithium, silicon, selenium; the spectrograph has shown infinitesimal quantities of gold, silver, lead, aluminium, tin, zinc, bismuth, cobalt, molybdenum, tungsten, gallium, germanium, palladium, glucinum, vanadium *et cetera*.

Some water contains microscopical algae (Glairines, Barélines).

Therefore, it can be seen that mineral waters are complex and strongly ionized solutions and suspensions. This is why so many are fragile and "living", and cannot be kept and bottled.

The strong and sometimes dangerous effect of mineral water on man is proved by innumerable clinical data, scientifically collated and studied. These studies enable us to know which patients should go to a certain spa and which ones should avoid it. Moreover, this century the effect of mineral waters has been studied by experimentation on man, animals, isolated organs and plants. All this research has been accomplished through the chairs of hydrology in medical schools, by laboratories of certain spas, for medical theses, and by means of special scholarships. It has demonstrated, among many other results, the fixation of thermal iron, arsenic and sulphur in the blood and liver, changes in metabolism, increase of vitamin C in tissues, activation or inhibition of diastases, protective action against microbial toxins and anti-allergic action. The inner mechanism of effect is, of course, far from being completely known, but it has been proved that there is a pharmacodynamic action, the water acting as a chemical drug, an effect on the autonomic nervous system and an effect on endocrine glands and on the enzymatic system of tissues. The matter remains still very complex, and crenotherapy can be considered above all as a medicine of "temperament and terrain".

Of course, many attempts have been made to make an artificial thermal water, use being made of the result of a complete analysis. The action of the "synthetic water", determined either clinically or experimentally, is extremely different from that of natural water, and generally very weak. Reasons given for this are the presence of elements in small quantity not detected in natural water, the "new-born conditions" of minerals, the electric condition, the colloidal state, the radioactivity, and the equilibrium of ions with synergic action in natural water.

Except for a few mineral waters that can be bottled and sent away, the patient must go to the springs.

Control and Protection of Mineral Water.

King Henry IV of France, at the end of the sixteenth century, introduced the first legislation regarding mineral water after he had seen both men and horses bathing in the same thermal bath or pool. Nowadays, very complete and extensive regulations control the use of springs, the hygienic conditions of thermal establishments and the constancy of the water's composition.

Every spring must obtain an "authorization for use" given by the Minister of Public Health after an exhaustive inquiry—that is, complete analysis of water, plan of projected thermal establishment, approval of Mines Department (geological aspect), Hygiene Department, Academy of Medicine, and *Institut national d'hydrologie et de climatologie*. The spa also applies for the protection of a large area of surrounding country to ensure that the water will not be polluted.

When in use a thermal spring or establishment is under the permanent control of the Mines Department on one hand, and of several governmental hygiene and medical authorities. Moreover, every year, the Prefect or government representative of the Province must check the results

of two bacteriological analyses of the water and a report on the establishment's activity during the year.

In provincial departments of hygiene and also at the Academy of Medicine in Paris there is a complete dossier for each thermal spring, where every analysis, report or other information since its "authorization for use" is kept. Thus every possible guarantee is given patients taking a thermal treatment.

Social Thermalism.

Social thermalism began officially with Henry IV, who ordered his "superintendents of fountains" to make the benefits of thermal water available to the pauper. The French Revolution decided that free travel and free accommodation would be provided for the poor in need of thermal treatment. Special free hospitals have existed for centuries in spas. Civil servants, returned soldiers and, as time went on, more and more groups of the populace received free thermal treatment. In 1936 the National Health Insurance began payments for children's treatments. After World War II the new National Health Insurance (Social Security), compulsory for every employee, introduced a liberal organization respecting the great principle of freedom of choice of physician and also of means of travel and accommodation.

The thermal treatment is prescribed by the practitioner. The patient must ask the agreement of the "Social Security", whose physicians then give their opinion on the utility of or indications for thermal treatment. If they disagree with the practitioner, the patient can ask the arbitration of a specialist in hydrology. When the "Social Security" has accepted the case, it pays, or rather reimburses, the patient 80%, or 100% in special cases, of the medical fees, and 80% or 100% of thermal establishment expenses, the price of the return ticket by train and a fixed indemnity for board and lodging.

It can be said today that French practitioners have in crenotherapy a valuable therapy against a number of diseases, and that a thermal cure is accessible to everyone.

Correspondence.

MEDICAL ASPECTS OF TATTOOING.

SIR: The surgical withers of all of us must have surely at one time or another been wrung when, yielding to the clamour of a "Malade imaginaire", we have done a forlorn-hope operation for "adhesions" or some such nebulous pathological condition and thereby riveted life-long fetters on our limbs.

In "Current Comment" (M. J. AUSTRALIA, August 21, 1954) you suggest that any outstanding "major opus" might well be signed "J. Smith fecit". To this I would add "provided the scar is in a place which might be reasonably inspected".

When "Waltzing Matilda" was a more sought after profession than it now is, its devotees were said to have a code of marks which they scrawled on gates and such like; for instance, one meant: "The Boss is a miserable Scotchman and has a pack of hungry savage dogs."

I suggest that a world-wide effort be made to register through some such body as U.N.O. a code of marks to be tattooed near the scar, and I suggest that the first should be a cross, which should mean: "Anybody opening this belly is as big a fool as I have been and deserves all that is coming to him."

Just think of the oceans of tears it would save . . . the surgeons.

Yours, etc.,

143 Macquarie Street,
Sydney,
August 23, 1954.

T. M. FURBER.

SIR: I was greatly interested in the article "Medical Aspects of Tattooing" in your issue of August 21. When holding the chair of physiology in the University of Melbourne I was on a few occasions consulted by male students who wished to have tattoo marks removed. The usual cause of this desire was that the young man had the name of an *amemorate* tattooed on arm or breast, but had subsequently transferred his affections and was embarrassed by this tell-tale evidence of a former attachment. I gave each the

option, a surgical operation with possible resultant scar or a little more tattooing, namely, a sailing ship, not too obsolete in design, with the letters "S.S." before the girl's name. The latter generally worked.

Yours, etc.,

Kangaroo Ground,
Victoria,
August 25, 1954.

W. A. OSBORNE.

THE ENIGMA OF THE MONA LISA SMILE.

SIR: Dr. Frank Trinca's article in THE MEDICAL JOURNAL OF AUSTRALIA of August 14, 1954, prompts the following comment.

There is a condition known as birth palsy, produced by a small subdural hematoma caused by a difficult birth, possibly a forceps delivery. Its pressure presumably damages a small area of the cortex concerned with motor impulses to the facial muscles on the opposite side. This may leave no trace, or may result—not in a paralysis—but in a permanent loss of tone of the muscles involved. This will not be apparent except to a keen observer, but only in a full-face photograph, when the face is in complete repose. The loss of tone of the facial muscles on the affected side results in an expression of apathy.

This will not be found in a painting, as the artist, if he is observant enough to notice it, would probably depict his sitter in a half-profile. It is very noticeable in the photograph of Arnold Bennett in the first volume of his "Diaries". It may be argued that his is a pronounced case, as he has a slight ptosis of the upper lid; but in lesser cases, where there is no ptosis, there will be noticed (in a photograph) a slight droop of the lower lid, making the eye look larger, and a less-marked oro-nasal crease or a just perceptible droop of the corner of the mouth. And if each side in turn is covered with a card, the contrast between the animation of the one side and the "dead-pan" appearance of the other cannot escape notice.

I need hardly say that such a condition in no way impairs the intellect, but it is by no means uncommon if one looks for it in the repose necessitated by a photograph.

Da Vinci, being a genius, possibly painted exactly what he saw, without glossing over what appeared to him as asymmetry. I have found this asymmetry in many photographs of celebrities and others, and I do not think it is due to any super-subtlety or "mystique", but to a slight birth trauma.

I humbly offer this as a possible explanation of the Mona Lisa smile, if indeed an explanation is called for. It is less recondite than Dr. Trinca's, whose metaphysical flights leave me far behind—gasping.

Yours, etc.,

E. TEMPLE SMITH.

Sydney,
August 23, 1954.

CONCERNING PROCTOLOGY.

SIR: Proctologists appear more prone than most surgeons to manifest the following: *furor scribendi*, *mania illustrationibus*, *stoliditas*, and *obsecutio* (asseveration with much calling on the gods). They ought to remember that almost alone in modern surgery they depend on pre-Listerian methods, employ mass ligation of pedicles with heavy non-absorbable seton-ligatures, regard "laudable" pus with equanimity and advocate healing by second intention.

Recent authors in this and other Australian surgical publications have deprecated (even threatening dire complications) any endeavours to bring proctology into line with modern surgical methods, by seeking primary closure and healing by first intention. With regard to hemorrhoids and fissures I know these asseverations to be false, for I have been using primary closure in these conditions for twelve years. Indeed, the freedom from pain, the short hospital stay, the absence of gauze and tubes in the anorectal canal, the abolition of post-operative digital dilatation and the ability to perform radical anal resection ("the Whitehead procedure") without stricture in leucoplakia and advanced hemorrhoids have been pleasing rewards for this enterprise. I have also been so rash as to indicate the circumstances under which primary closure in fistulous conditions might be undertaken (Starr, 1953).

This constitutes the majority of perianal and, with experience, an increasing number of ischio-rectal fistulae. I was encouraged to this surgical temerity because of the number of cases treated by orthodox methods who had residual disabilities. These latter included recurrence, discharge, and pseudo-incontinence (leakage of mucus without faeces) and appeared to be associated with a deep epithelialized cleft plunging between the divided ends of the external sphincter and forming a perianal cesspool. Dealing with these cases by primary closure has provided a ready solution to a difficult and perplexing problem.

For the present, proctology tends to lag indolent upon the surgical stage. Its shortcomings should be critically examined in the light of modern surgical techniques. Modernization of its surgical philosophy will inevitably follow.

Yours, etc.,

149 Macquarie Street,
Sydney,
August 20, 1954.

KENNETH W. STARR.

Reference.

STARR, K. W. (1953), *Postgraduate Medicine*, 14: 365.

Naval, Military and Air Force.

APPOINTMENTS.

The following appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 49, of August 12, 1954.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Appointments.—James Crichton, Brian Tremayne Treloar and Robert Arthur Franklin are appointed Surgeon

Lieutenants (for short service) (on probation), dated 19th May, 1954, 2nd June, 1954, and 15th June, 1954, respectively.

Termination of Appointment.—The appointment of James Crichton as Surgeon Lieutenant (for short service) (on probation) is terminated, dated 28th June, 1954.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Appointments.—Martin Desmond Begley is appointed Surgeon Lieutenant, with seniority in rank of 3rd January, 1950, dated April, 1954. George Robert Faithfull is appointed Surgeon Lieutenant, dated 28th May, 1954.

AUSTRALIAN MILITARY FORCES.

Citizen Military Forces.

Western Command: Fifth Military District.

Royal Australian Army Medical Corps (Medical).—5/32054 Colonel J. H. Stubbe, E.D., relinquishes the appointment of Deputy Director of Medical Services, Headquarters, Western Command, 31st July, 1954, is placed upon the Retired List (5th Military District), and is granted the honorary rank of Brigadier, with permission to wear the prescribed uniform, 1st August, 1954. 5/26391 Lieutenant-Colonel R. R. Anderson, M.C., is absorbed in a vacancy within the authorized establishment of Lieutenant-Colonels, and to receive pay and allowances of that rank, 1st August, 1954. 5/26391 Lieutenant-Colonel R. R. Anderson, M.C., is appointed Deputy Director of Medical Services, Headquarters, Western Command, and to be Colonel, 1st August, 1954.—(Ex. Min. No. 145—Approved 3rd August, 1954.)

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

The short-service commission of Flight Lieutenant (Acting Squadron Leader) A. D. Litchfield (024304) is extended for a period of three years, 12th June, 1955.

The resignation of Flight Lieutenant F. I. Walke (012787) is accepted, 30th June, 1954.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED AUGUST 21, 1954.¹

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	6(1)	..	4(3)	..	4(3)	14
Amoebiasis	1(1)	1	..	2
Ancylostomiasis
Anthrax
Bilharziasis
Brucellosis	1(1)	1(1)	2
Cholera	1(1)	1	2
Chorea (St. Vitus)
Dengue
Diarrhoea (Infantile)	1(1)	13(11)	9(9)	23
Diphtheria	3(1)	1	1(1)	..	5(3)	10
Dysentery (Bacillary)	3(3)	3
Encephalitis	2(2)	2
Filariasis
Homologous Serum Jaundice
Hydatid
Infective Hepatitis	54(28)	17(7)	5(5)	8	79
Lead Poisoning	1	..	1	2
Leprosy
Leptospirosis	1	..	5(1)	6
Malaria
Meningococcal Infection	7(4)	..	3(2)	..	1	1	12
Ophthalmia
Ornithosis
Paratyphoid
Plague
Poliomyelitis	2(2)	13(5)	2	2(1)	1(1)	20
Puerperal Fever	2(2)	2
Rubella	2(1)	11(11)	13
Salmonella Infection
Scarlet Fever	15(8)	27(9)	5(2)	3(2)	7(7)	1	58
Smallpox
Tetanus	1(1)	1
Trachoma	12	12
Trichinosis
Tuberculosis	41(24)	16(12)	11(7)	7(5)	11(7)	5(3)	61
Typhoid Fever	1(1)	..	1	1(1)	3
Typhus (Flea-, Mite- and Tick-borne)	2	2
Typhus (Louse-borne)
Yellow Fever

¹ Figures in parentheses are those for the metropolitan area.

Active Citizen Air Force.**Medical Branch.**

No. 21 (City of Melbourne) Squadron.—Squadron Leader A. M. Beech (033454) is transferred to the Reserve, 30th June, 1954.

Australian Medical Board Proceedings.**QUEENSLAND.**

THE following has been registered, pursuant to the provisions of the Medical Acts, 1939-1948, as a duly qualified medical practitioner: Reid, James Campbell, M.B., Ch.B., 1932 (Univ. Glasgow), F.R.C.S. (Ireland), 1951.

The following additional qualifications have been registered: Ure, James Noel, D.C.H. (London), 1954; Bennett, Roger Alwin, D.A., R.C.P. and S. (England), 1954.

TASMANIA.

THE following has been registered, pursuant to the provisions of the Medical Act, 1918, as a duly qualified medical practitioner: Hatherley, Mona Elizabeth, M.B., Ch.B., 1923 (Univ. Sheffield).

The following additional qualification has been registered: Lewis, Reginald Abbott, F.F.A. (R.A.C.S.).

Notice.**AUSTRALIAN ASSOCIATION OF CLINICAL PATHOLOGISTS.**

THE annual general meeting of the Australian Association of Clinical Pathologists will be held in Tasmania (partly in Launceston and partly in Hobart) from October 21 to 23, 1954. Papers will be contributed by Dr. J. E. McCartney ("The Use of the Microscope in Clinical Pathology"), Dr. M. P. K. Shoobridge, Dr. J. V. Duhig ("Bacterial Sensitivity to Antibodies"), Dr. J. E. Bonnin ("Erythrophagocytosis in Vitro"), Dr. C. A. Duncan ("Bronchiolar Carcinoma"), Dr. M. Fowler ("Staphylococci and Renal Infarction"), and Dr. R. L. Quinn ("Cytomegalic Inclusion Disease"). A general discussion will be held on "Cytodiagnosis". The honorary secretary of the Association is Dr. R. L. Quinn, c/o Medical School, Herston Road, Brisbane, Queensland.

Medical Appointments.

Dr. Bogoslaw Grigoroff has been appointed quarantine officer at Wyndham.

Dr. Brian Walsh has been appointed quarantine officer at Derby.

Dr. F. M. Hooper has been appointed honorary assistant radiologist to the Royal Alexandra Hospital for Children, Sydney.

Dr. T. H. O'Donnell has been appointed honorary assistant ear, nose and throat surgeon to the Royal Alexandra Hospital for Children, Sydney.

Dr. R. E. Dunn has been appointed honorary relieving assistant ear, nose and throat surgeon to the Royal Alexandra Hospital for Children, Sydney.

Dr. C. A. Sara has been appointed visiting anaesthetist to the Royal Alexandra Hospital for Children, Sydney.

Dr. E. B. Tunbridge has been appointed a medical officer in the Department of Public Health, Tasmania.

Deaths.

THE following death has been announced:

MCMAHON.—Frank Fitzroy McMahon, on August 25, 1954, at Lilydale, Victoria.

Diary for the Month.

- SEPT. 13.—Victorian Branch, B.M.A.: Finance Subcommittee.
SEPT. 14.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 15.—Western Australian Branch, B.M.A.: General Meeting.
SEPT. 16.—Victorian Branch, B.M.A.: Executive of Branch Council.
SEPT. 21.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 22.—Victorian Branch, B.M.A.: Branch Council Meeting.
SEPT. 23.—New South Wales Branch, B.M.A.: Clinical Meeting.
SEPT. 24.—Queensland Branch, B.M.A.: Council Meeting.
SEPT. 28.—New South Wales Branch, B.M.A.: Ethics Committee.
SEPT. 30.—New South Wales Branch, B.M.A.: Branch Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute: Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Tasmania: Part-time specialist appointments for the north-west coast of Tasmania.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognise any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 per annum within Australia and the British Commonwealth of Nations, and £6 10s. per annum within America and foreign countries, payable in advance.